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INFORMATION REPORT

REPORT NO [redacted]

CD NO.

CASE [redacted] 25X1A

COUNTRY China/Czechoslovakia/Poland/USSR

DATE DISTR. 29 Jan 1951

SUBJECT Subscribers to Transactions American Academy of Ophthalmology and Otolaryngology

NO. OF PAGES 1

PLACE ACQUIRED - - - - - - 25X1A

NO. OF ENCLS. (LISTED BELOW)

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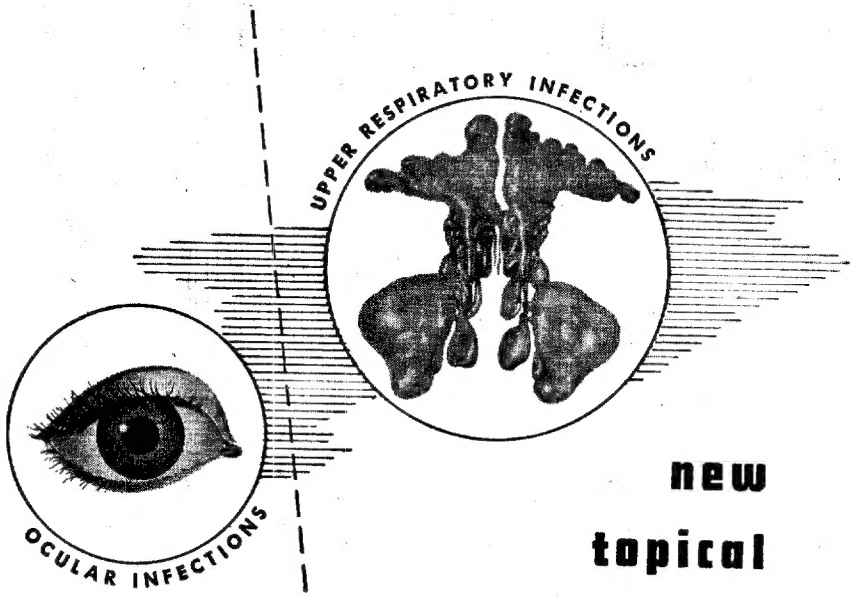
TRANSACTIONS

AMERICAN ACADEMY
of
OPHTHALMOLOGY
and
OTOLARYNGOLOGY

NOVEMBER - DECEMBER
1950

Printed bimonthly, February, April, June, August, October, and December, by the Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska, for the American Academy of Ophthalmology and Otolaryngology. Editorial and business office, 100 First Avenue Building, Rochester, Minnesota. Subscription price $5.00 yearly to members, $10.00 yearly to non-members. Entered as Second-Class Matter November 28, 1938, at the Post Office at Omaha, Neb., under Act of March 8, 1879.

ANNUAL MEETING OCTOBER 14-19, 1951, CHICAGO



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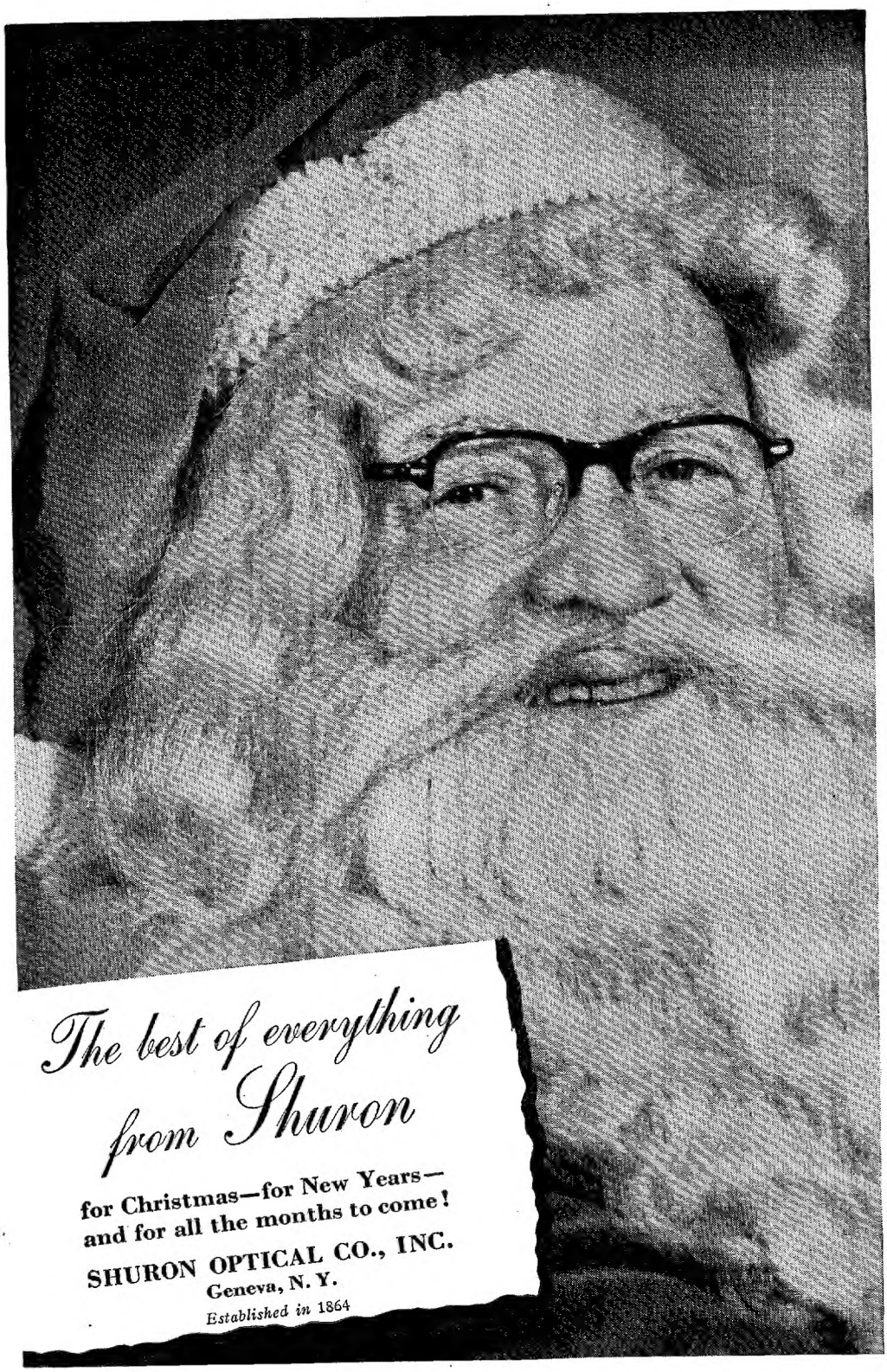
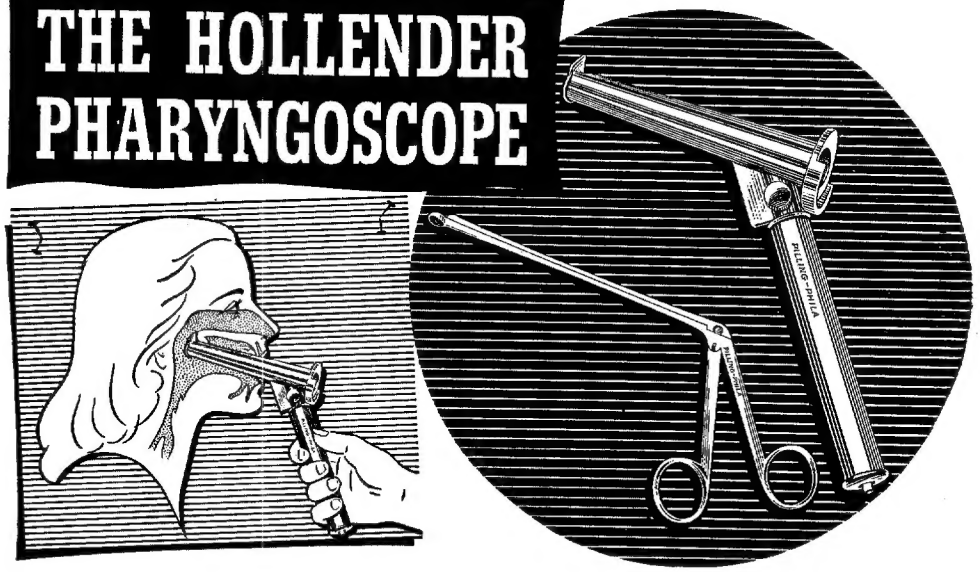


Authentic Instruments for Otorhinolaryngology

THE HOLLENDER PHARYNGOSCOPE



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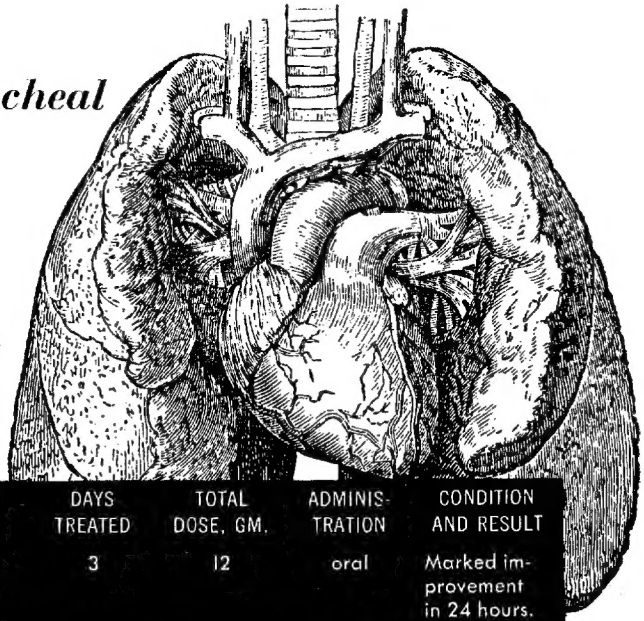
in acute laryngotracheal bronchitis prompt response with Terramycin



CASE	DIAGNOSIS	INFECTING ORGANISM	DAYS TREATED	TOTAL DOSE, GM.	ADMINISTRATION	CONDITION AND RESULT
16	Acute laryngotracheal bronchitis	Hemophilus Influenzae	3	12	oral	Marked improvement in 24 hours. Recovery

Case report taken from Herrell, W. E.; Heilman, F. R., and Wellman, W. E.: Ann. New York Acad. Sc. 53:448 (Sept. 15) 1950.

CRYSTALLINE

Terramycin

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Dosage: On the basis of findings obtained in over 150 leading medical research centers, 2 Gm. daily by mouth in divided doses q. 6 h. is suggested for most acute infections.

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100 mg. capsules, bottles of 25 and 100;
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1. Welch, H.; Hendricks, F. D.; Price, C. W., and Randall, W. A.: J. A. Ph. A. (Sc. Ed.) 39:185 (Apr.) 1950.
2. Knight, V.: New York State J. Med. 50:2173 (Sept. 15) 1950.

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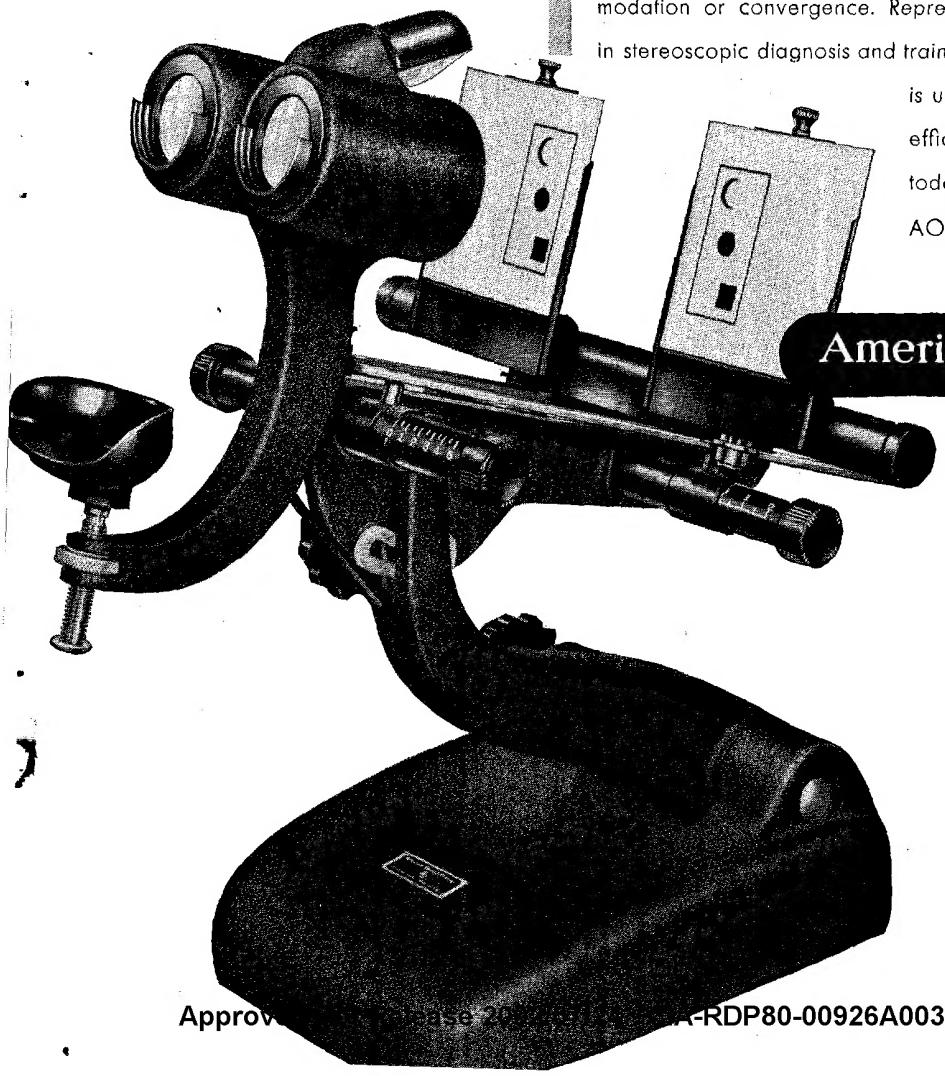

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. . . the Campimeter that permits complete plotting of both central and paracentral fields under stereoscopic fixation

In many cases showing impairment of vision it is important to make full central and paracentral field studies. The "angled mirror septum" in the AO Lloyd Stereo Campimeter permits field testing up to 47° nasally, 35° temporally, 30° upward, and 30° downward. Even when central scotomata are present in either or both eyes, single binocular fixation is readily maintained. Lenses compensate for the distance from the chart to the eye, thus avoiding accommodation on the part of the patient. Illumination of test chart and fixation chart is exceptionally even.

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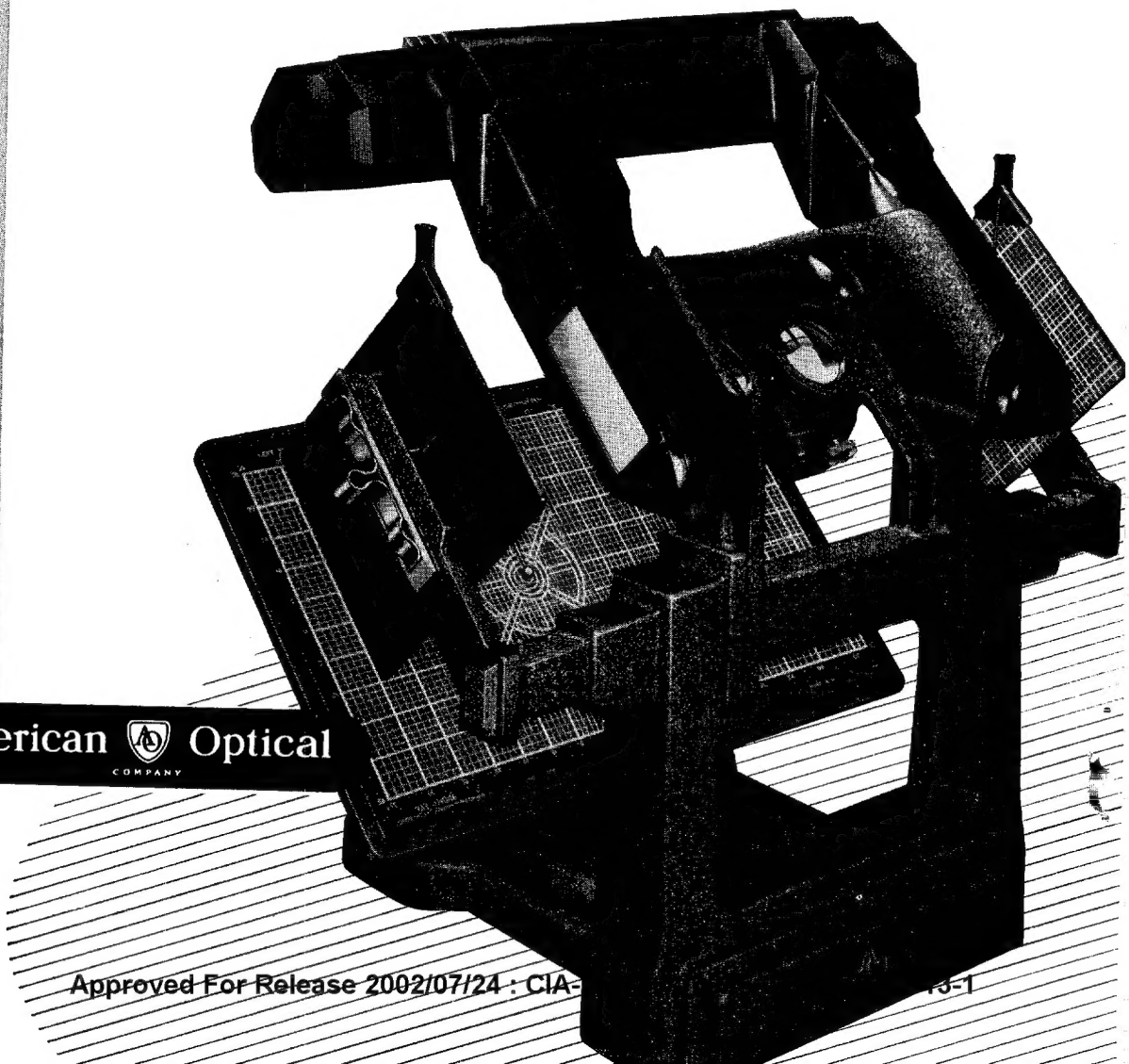

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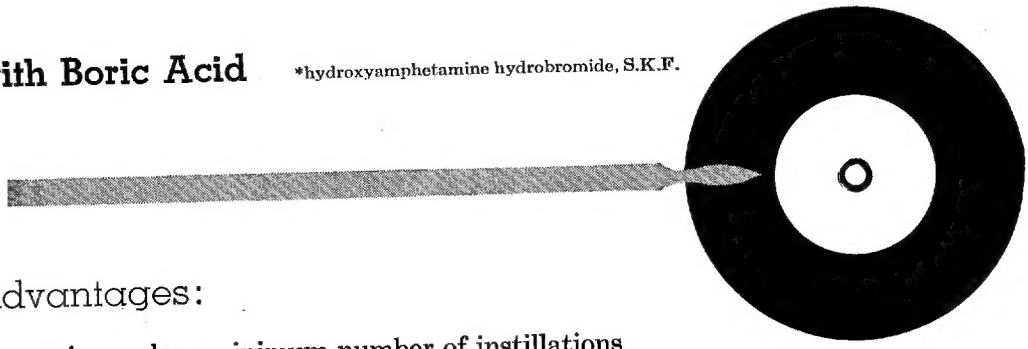

Advantages:

1. requires only a minimum number of instillations
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3. permits accurate refraction
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The 'Paredrine Technique' in refraction saves the physician time and trouble and spares the patient prolonged visual disability. In the words of Harrison[1], it "has the advantage of maximum action with minimum duration." With children, the whole procedure can be carried out at the office; and the greatly reduced amount of atropine required for satisfactory cycloplegia lessens the probability of toxic side effects.

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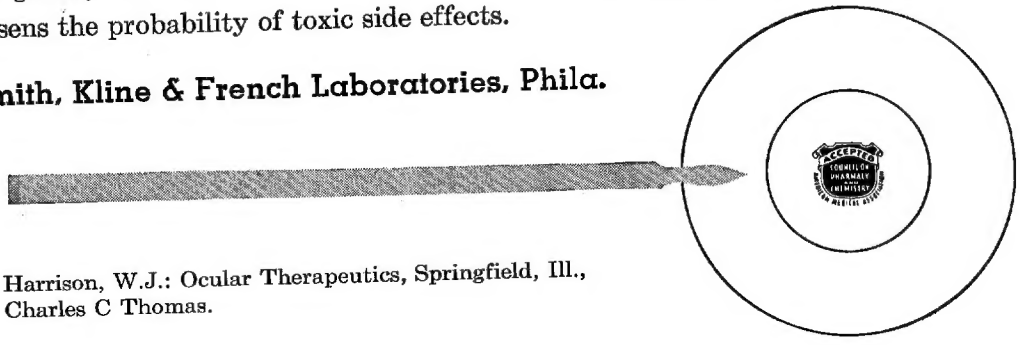

1. Harrison, W.J.: Ocular Therapeutics, Springfield, Ill., Charles C Thomas.

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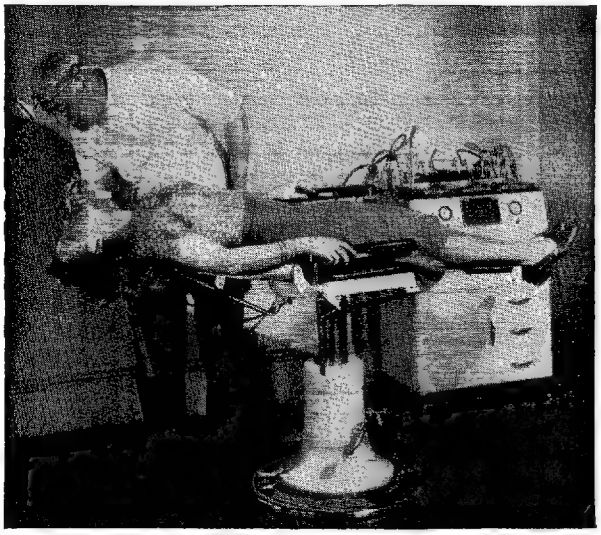

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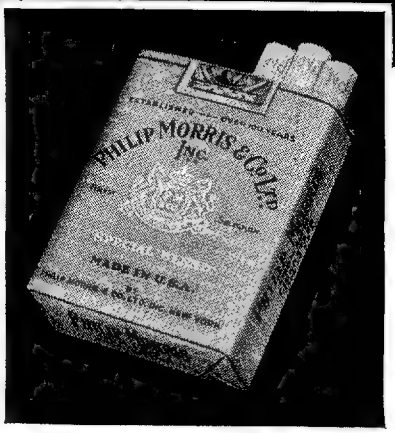

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**Proc. Soc. Exp. Biol. and Med.*, 1934, 32, 241-245; *N. Y. State Journ. Med.*, *Vol.* 35, 6-1-35, *No.* 11, 590-592, *Laryngoscope, Feb.* 1935, *Vol. XLV, No.* 2, 149-154; *Laryngoscope, Jan.* 1937, *Vol. XLVII, No.* 1, 58-60

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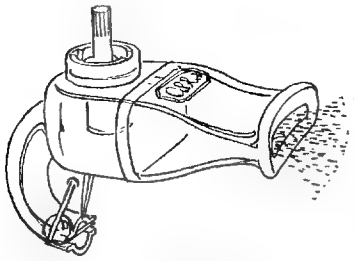

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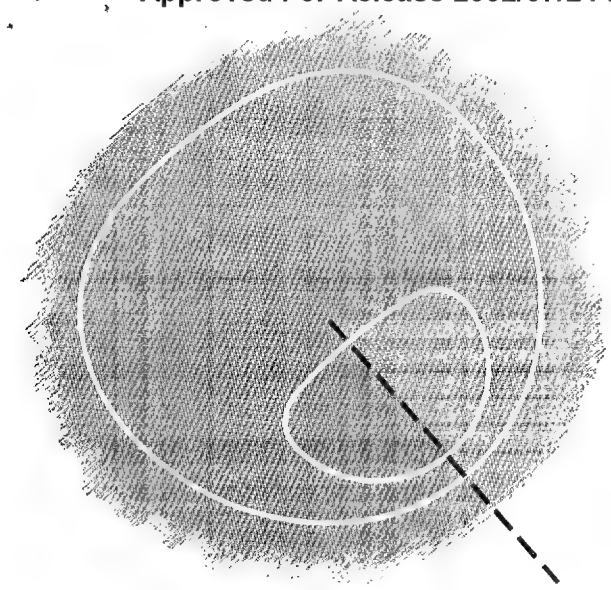
1. Krasno, L. R., Grossman, M. I., and Ivy, A. C. (1949). The Inhalation of 1-(3',4'-Dihydroxyphenyl)-2-Isopropylaminoethanol (Norisodrine Sulfate Dust), J. Allergy, 20:111, March. 2. Krasno, L. R., Grossman, M., and Ivy, A. C. (1948). The Inhalation of Norisodrine Sulfate Dust, Science, 108:476, Oct. 29.



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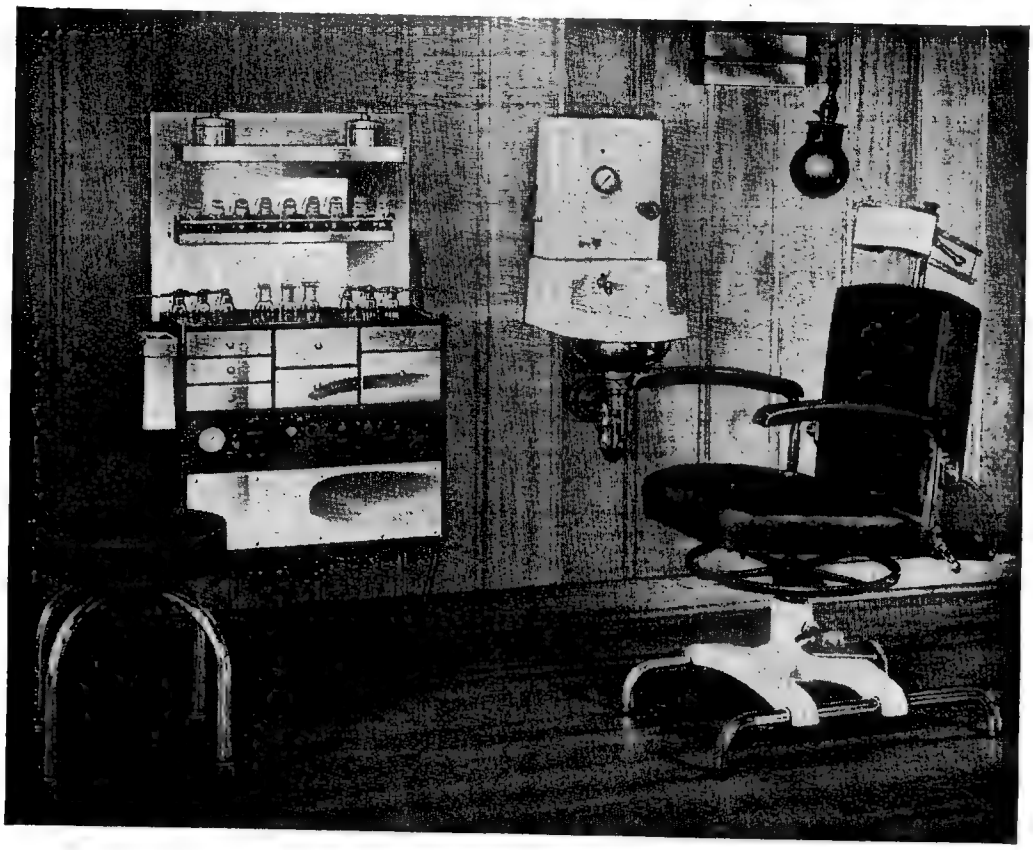
PANOPTIK
is the bifocal lens which provides the widest possible reading area with least encroachment on distance vision

BAUSCH & LOMB *Orthogon Panoptik*



A COMPLETE TREATMENT ROOM IN A SMALL AREA

The dimensions in the above photograph are 5' 9" along one wall and 4' 2" along the other. In this area, conveniently arranged, is everything needed to make a complete examination. However, it need not be confined to this limited space. The complete absence of piping and wiring is especially noticeable. A number of other arrangements will be furnished on request.

Treatment Stand: Covered with stainless steel. Contains: air regulator with gauge, tubing, cutoff and air filter; nasopharyngoscope rheostat; push button; cautery transformer and rheostat; transilluminator complete with rheostat, cord, handle, lamps, antrum and frontal tips; seven metal drawers on fiber runners and one large drawer for towels; waste container; bakelite covered work table; space for six spray bottles, twelve solution bottles, shelf for cotton jars; pocket for history card or prescription blanks; and a spray bottle heater; the whole presents an attractive appearance and will not tarnish and is easy to keep clean. Price complete without glassware $185.00. Without air but with electrical equipment $155.00. With air equipment only $140.00. Without either air or electrical equipment $110.00.

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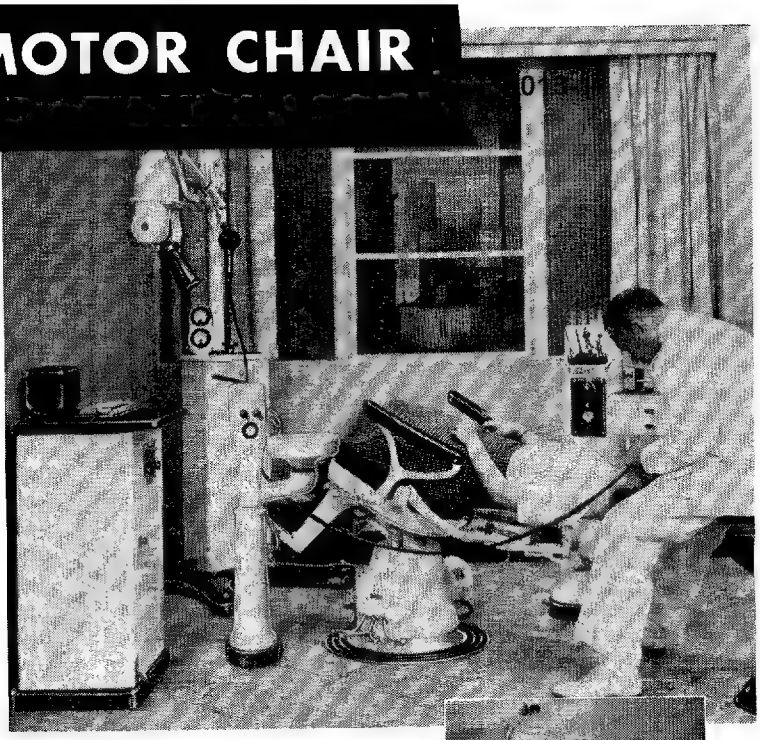
for rapid easy adjustment to any treatment position



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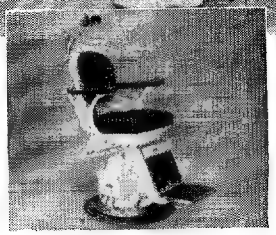
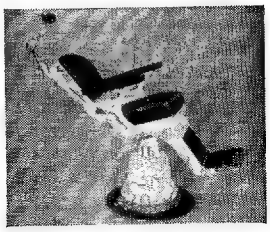
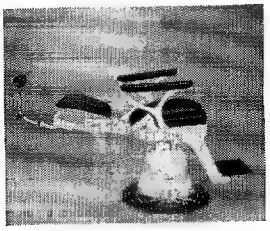


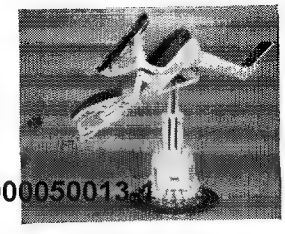
Slight pressure on levers . . .



. . . instantly positions Ritter ENT Motor Chair . . .



. . . at any desired angle . . .



. . . from sitting to supine.

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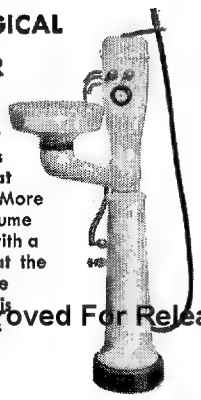
A Ritter Surgical Cuspidor, completely redesigned, provides greater efficiency at low water pressure. More effective vacuum volume control maintained with a fingertip regulator at the aspirator handle. Use any aspirator tip. This modern equipment is a necessity in every doctor's office.



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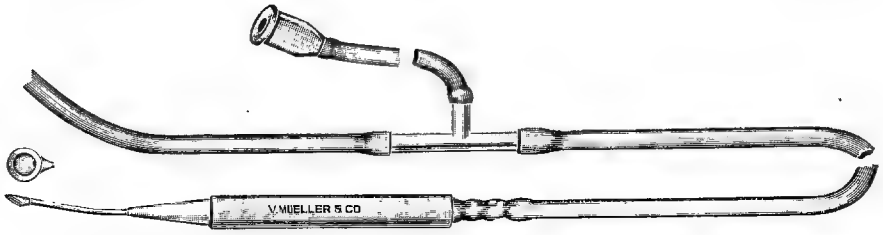

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A-14 — This improved erisophake permits greater ease of application and more perfect manipulation in extraction procedures than formerly. It makes use of the Dimitry shallow suction cup, and has Nugent's oral vacuum control valve—most simple, efficient means of control of vacuum during cataract extraction. Handpiece and oral valve are gold plated. Erisophake, valve and tubing only.. **$24.75**

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WIDE ANGLE VIEW

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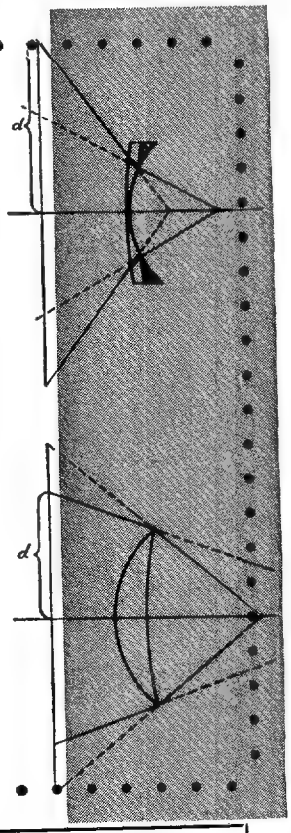

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SCIENTIFIC PAPERS READ AT
FIFTY-FIFTH ANNUAL SESSION

CHICAGO, ILLINOIS
October 8-13, 1950

PRINTED IN OMAHA, NEBRASKA
BY
DOUGLAS PRINTING COMPANY

PRINTED IN U.S.A.

American Academy of Ophthalmology and Otolaryngology

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THE OCULAR FINDINGS OF INTRACRANIAL TUMOR

A Study of 358 Cases

MANOUSOS ANGEL PETROHELOS, M.D.
BY INVITATION
and
JOHN WOODWORTH HENDERSON, M.D.
ANN ARBOR, MICH.

THE diagnosis and successful localization of intracranial neoplasms should ideally be the result of close cooperation between the neurosurgeon, the neurologist and the ophthalmologist. Such a relationship has been enjoyed at the University Hospital of the University of Michigan, where the neurosurgery and neurology departments routinely refer the great majority of their suspected cases of intracranial pathology for ophthalmologic consultation. There has thus been afforded a somewhat unique opportunity to establish a proper background upon which the neurologist or neurosurgeon may rely in reaching his diagnosis.

It is the purpose of this paper to attempt an evaluation of the signs of intracranial neoplasm which are encountered by the ophthalmologist from the standpoint of their significance in diagnosis.

A total of 358 cases will be presented. Of these 344 were operated upon and the pathologic diagnosis confirmed by microscopic study. The remaining 14 cases were those in which the diagnosis was within the limits of certainty but in which it was felt surgery would not be advantageous. The latter instances fell mainly under the heading of pinealoma. The original series of records was selected by the Division of Medical Statistics to include representative series of each major diagnostic group, either by location or by type of tumor. These were tabulated according to ophthalmologic findings. The individual findings were then analyzed according to tumor location, as will be noted from the accompanying tables. Thus the series presented does not claim to embrace the total number of cases observed during a given period of time, nor does it attempt to include all the cases of any one tumor type during such an interval. Practically all of the records studied were of patients seen within the past ten years. Where a particular finding was not noted for a tumor location, it has been omitted from the appropriate table.

The final selection of cases whose completeness warranted their inclusion in this study is presented in table I according to location of the tumor. Those instances listed as "diffuse" involvement were cases in which the tumor involved more than two major areas. The remaining subdivisions of the table are self explanatory. This grouping does not claim any significance with regard to the relative incidence of brain tumor by local sites.

The ocular findings to be analyzed are (1) papilledema, (2) optic atrophy, (3) visual field changes, (4) pupillary abnormalities, and (5) disorders of ocular motility.

From the department of ophthalmology, University of Michigan Medical School. Dr. Petrohelos holds the position of John E. Weeks Scholar in Ophthalmology, and Dr. Henderson that of Walter R. Parker Scholar in Ophthalmology.

Presented at the Fifty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 8-13, 1950, Chicago, Ill.

TABLE I

PAPILLEDEMA

TUMOR LOCATION	NUMBER OF CASES	NUMBER WITH PAPILLEDEMA	PERCENTAGE	BILATERAL	UNILATERAL
Frontal lobe	66	30	45.4	29	1
Temporal lobe	29	15	51.7	15	—
Parietal lobe	25	13	52.0	13	—
Occipital lobe	5	4	80.0	4	—
Frontoparietal	18	9	50.0	7	2
Temporoparietal	17	10	58.8	10	—
Parieto-occipital	11	9	81.8	9	—
Frontotemporal	7	3	42.9	3	—
Cerebellum	58	44	76.2	44	—
Pineal gland	16	10	62.5	10	—
Third ventricle	7	5	71.0	5	—
Lateral ventricle	9	7	77.7	6	1
Fourth ventricle	16	12	75.0	12	—
Pons and medulla	6	5	83.3	5	—
Midbrain and pons	10	6	60.0	5	1
Posterior fossa	5	4	80.0	4	—
Corpus callosum	5	2	40.0	2	—
Basal ganglia	5	3	60.0	3	—
Cerebellopontine angle	6	5	83.3	5	—
Diffuse	10	7	70.0	7	—
Optic chiasm and nerve	9	5	55.5	3	2
Pituitary and craniopharyngioma	11	1	9.1	1	—
Sphenoidal wing meningioma	7	4	57.1	3	1
Totals	358	213	59.5	205	8

TABLE II

OPTIC ATROPHY

LOCATION OF TUMOR	PRIMARY ATROPHY BILATERAL	PRIMARY ATROPHY UNILATERAL	SECONDARY ATROPHY BILATERAL	SECONDARY ATROPHY UNILATERAL	TOTAL
Frontal	2	—	5	—	7
Occipital	—	—	1	—	1
Frontoparietal	—	2*	—	—	2
Cerebellum	1	—	6	—	7
Pineal	1	—	5	—	6
Third ventricle	—	—	1	—	1
Lateral ventricle	—	1†	1	1	3
Fourth ventricle	—	—	2	—	2
Basal ganglia	—	—	1	—	1
Diffuse	—	—	1	—	1
Optic chiasm and nerve	2	2	—	—	4
Sphenoidal wing meningioma	—	1*	—	—	1
Pituitary and craniopharyngioma	3	1	—	—	4
Midbrain and pons	1	—	—	—	1
Cerebellopontine angle	—	—	1	—	1
Totals	10	7	24	1	42

* Foster Kennedy syndrome

† ? Early Foster Kennedy syndrome

TABLE III

VISUAL FIELD FINDINGS

LOCATION OF TUMOR	FIELDS DONE	HOMONYMOUS HEMIANOPSIA	MACULA SPLIT	MACULA SPARED	BITEMPORAL HEMIANOPSIA	QUADRANTANOPSIA	PERIPHERAL CONSTRICTION	CENTRAL SCOTOMA	BINASAL HEMIANOPSIA
Frontal	40	2	—	1	—	—	7	1	—
Temporal	16	9	—	5	—	1	1	—	—
Parietal	22	5	—	—	—	—	1	—	—
Occipital	5	3	—	2	—	1	—	—	—
Frontoparietal	14	1	—	1	—	1	1	—	—
Temporoparietal	9	5	—	2	—	—	—	—	—
Parieto-occipital	8	4	—	2	—	2	—	—	—
Frontotemporal	5	—	—	—	—	—	1	—	—
Cerebellum	23	1	1	—	—	—	1	—	—
Pineal gland	10	1	—	—	—	—	2	—	—
Third ventricle	4	—	—	—	—	1	—	—	—
Lateral ventricle	7	1	1	—	1	1	4	—	—
Fourth ventricle	10	—	—	—	—	—	3	—	—
Pons and medulla	6	—	—	—	—	—	1	—	—
Midbrain and pons	8	—	—	—	—	—	2	—	—
Posterior fossa	3	—	—	—	—	—	—	—	—
Corpus callosum	5	1	—	—	—	—	—	—	—
Basal ganglia	2	—	—	—	—	—	—	—	—
Cerebellopontine angle	4	—	—	—	—	—	1	—	—
Diffuse	7	2	1	—	—	—	1	1	—
Optic chiasm and nerve	8	—	—	—	3	—	1	—	2 (unilateral)
Pituitary and craniopharyngioma	9	—	—	—	8	—	—	—	—
Sphenoidal wing meningioma	6	—	—	—	—	—	1	—	—
Totals:	231	35	3	13	12	7	28	2	2

The incidence of papilledema was 59.5 per cent, this finding being reported in 213 of the 358 cases. Where the tumors could be classified as located above the tentorium, papilledema was evident in 53.3 per cent (137 out of a total of 257 cases). Those located subtentorially showed papilledema in 76 out of 101 cases, an incidence of 75.2 per cent. Papilledema was reported to be bilateral in 205 of the 213 individuals. In only 14 cases was there a difference in amount of elevation of the nerve head between the two eyes sufficient to warrant its mention in the hospital records.

The incidence of optic atrophy was 12 per cent among the cases studied. There were 25 instances in which the optic nerve changes were classified as secondary atrophy. Those diagnosed as primary optic atrophy totaled 17 cases. In both types of atrophy the greater number showed changes in both eyes. The findings by location of the tumor are given in table II.

The visual field findings are listed in table III. A visual field examination was performed in 231 of the 358 cases. Changes in the fields of vision were recorded in 104 of the 231 examinations, or in slightly less than half. The specific findings for each group of tumors may be noted in the table. Almost without exception there were positive visual field findings in certain cases regardless of tumor location. Although too few

cases were found to warrant inclusion in the table, the incidence of improvement of the visual fields after surgery was small. It was noted according to further perimetry or to report in the records in only 3 cases. The poor prognosis of many of the patients undoubtedly is reflected in this finding.

Anisocoria was the only pupillary abnormality thought reliable enough for inclusion in this report. This was mentioned in 42 of the records, an incidence of 12 per cent. The lateralization of the pupillary size was unfortunately not mentioned in many cases. The larger pupil was on the same side as the tumor in 13 instances, as compared with 6 cases in which it was contralateral to the lesion. Table IV lists the cases in detail.

TABLE IV

ANISOCORIA

LOCATION OF TUMOR	ANISOCORIA PRESENT	LARGER ON SAME SIDE	LARGER ON OPPOSITE SIDE
Frontal	7	4	2
Temporal	8	6	2
Occipital	1	—	—
Temporoparietal	3	2	1
Frontotemporal	1	—	1
Cerebellum	9	—	—
Third ventricle	1	—	—
Fourth ventricle	2	—	—
Pons and medulla	3	—	—
Corpus callosum	2	—	—
Basal ganglia	1	—	—
Cerebellopontine angle	1	1	—
Diffuse	1	—	—
Pituitary and craniopharyngioma	1	—	—
Sphenoidal wing meningioma	1	—	—
Totals	42	13	6

The movements of the eyes were found to be affected in a fairly large number of the cases studied. Defects of conjugate movement were noted in 20 cases of the group. These are listed in table V. Most of these instances occurred in patients in whom the location of the tumor was such as to involve the pathways for ocular movement, either in the frontal lobe or in the brain stem, with particular emphasis upon the group of pineal tumors.

A lack of uniformity of classification as well as inadequate description of particular cases in detail makes the evaluation of nystagmus in this series most difficult. However, it will be

TABLE V

CONJUGATE MOVEMENT DEFECTS

LOCATION OF TUMOR	UPWARD GAZE PALSY	LATERAL GAZE PALSY	MISCELLANEOUS	TOTAL CASES
Frontal	2	1	—	3
Temporal	1	—	1 (weakness all directions)	2
Cerebellum	1	—	—	1
Pineal	8	4*	—	8
Diffuse	1	—	—	1
Optic chiasm and nerve	1	—	—	1
Pituitary and craniopharyngioma	—	1	—	1
Sphenoid wing meningioma	—	1	—	1
Midbrain and pons	1	—	1 (upward and downward gaze)	2
Totals	15	7	2	20

* In 4 of the 8 cases listed.

TABLE VI

NYSTAGMUS

LOCATION OF TUMOR	VERTICAL	HORIZONTAL	MIXED HORIZONTAL AND VERTICAL	MISCELLANEOUS	TOTAL
Frontal	—	3	—	1 "central"	4
Temporal	—	1	—	—	1
Occipital	—	1	—	—	1
Cerebellum	1	11	7	1 "ocular"	20
Pineal	1	1	—	—	2
Lateral ventricle	—	1	—	—	1
Fourth ventricle	—	3	1	—	4
Pons and medulla	—	3	—	—	3
Diffuse	—	—	1	—	1
Cerebellopontine angle	—	—	2	—	2
Totals	2	24	11	2	39

noted from table VI that the majority of the cases were those in which coordinative mechanism of the cerebellum, the brain stem, or the motor pathways was likely to be involved. The incidence of nystagmus for the series was 10.9 per cent.

Individual muscle palsies occurred in 46 cases of the group. The abducens nerve or nerves were involved in 35, the oculomotor partially affected in 11, and the trochlear in only 2 patients. Several other cases could not be listed in this way. These included paralysis of convergence and complete ophthalmoplegias and may be found in table VII.

TABLE VII

EXTRAOCULAR MUSCLE PALSIES

LOCATION OF TUMOR	6TH PALSY	3RD PALSY	4TH PALSY	MISCELLANEOUS	TOTAL CASES
Frontal	2	1 inc.	—	1 ext. ophthalmoplegia O.U.	3
Temporal	2	1 compl.	—	1 compl. ophthalmoplegia O.U.	3
Frontoparietal	1	—	—	—	1
Temporoparietal	2	1 inc.	—	—	3
Cerebellum	8	5 inc.	—	2 convergence palsy	13
Pineal	5	1 inc.	—	—	6
Lateral ventricle	1	—	—	—	1
Fourth ventricle	2	1 inc.	—	1 ? convergence palsy	3
Pons and medulla	3	—	—	—	3
Midbrain and pons	1	—	—	2 convergence palsy	1
Basal ganglia	2	1	1	—	4
Diffuse	2	—	—	—	2
Optic nerve and chiasm	2	—	—	—	2
Pituitary and craniopharyngioma	1	—	1	—	2
Sphenoidal wing meningioma	1	—	—	—	1
Totals	35	11	2		48

DISCUSSION

The incidence of papilledema found for this series of cases is distinctly lower than that reported in other articles. Van Wagenen[8] found in a series of 145 verified cases of intracranial tumor that over 88 per cent developed papilledema. Critchley[1] has reported brain tumor to be associated with papilledema in over 80 per cent of cases. The 59.5 per cent incidence in the present series seems low by comparison. Truly comparable figures are not available, since the higher figures are given for series of tumors in children, or for consecutive cases over a relatively short period of time. The closest comparison could be made with the report of Paton,[6] who found that papilledema occurred in 80 per cent of intracranial tumors in a series of 252 cases.

The discrepancy between the findings of the present series and those of previous articles appears to be a direct reflection of the remarkable advances made in the diagnosis and treatment of brain tumors during the past several decades. The reports cited above all appeared prior to the period covered by the cases herein reported. With the diagnostic aids of electroencephalography, ventriculography, and arteriography, fewer cases of intracranial tumor are now allowed to progress to the stage where papilledema becomes visible to the ophthalmoscope.

The higher percentage (75.2 per cent) in the present report for tumors arising in the posterior fossa of the skull is much nearer to comparable figures in the literature. The papers of Critchley,[1] of Newman,[5] and of others report a high incidence of papilledema for tumors of the brain in children. It is true that the greater percentage of brain tumors in childhood are of the subtentorial variety. This closer correlation of reports for papilledema between subtentorial tumors for different age groups appears to reflect the earlier and more severe interference with the circulation of the cerebrospinal fluid with resulting internal hydrocephalus.

The preponderance of bilateral papilledema in this series serves to verify the statement of Duke-Elder,[3] who writes that in the great majority of cases the degree of edema is equal in the two eyes and that a greater amount of swelling in one optic nerve head is not of lateralizing value.

As would be expected, the majority of the cases of primary optic atrophy occurred where the direct action of the tumor was exerted upon the optic nerve, the optic chiasm or the optic tract. At least 13 out of the 17 instances of such atrophy could be so classified if one were to include the frontal, frontoparietal, optic chiasm and nerve, sphenoidal wing meningioma, and pituitary groups. All of the tumors so listed are also much less likely to produce papilledema until very late in their course. Nineteen of the 25 instances of secondary optic atrophy occurred in cases in which the ventricular system could be easily involved, and thus papilledema occurs early, to be followed by a post-papilledema type of atrophic change. The small number of cases evidencing the Foster Kennedy syndrome should be mentioned. None occurred in purely frontal lobe lesions, but two instances were found in more extensive tumor involvements classified as frontoparietal. The third case was found with a sphenoidal wing meningioma. The fourth instance was equivocal and occurred in a case with a tumor of the lateral ventricle.

It will be seen from table III that the most frequent visual field findings were homonymous hemianopsia and peripheral constriction. There were 35 instances of homonymous hemianopsia in the 231 examinations performed, or slightly over 15 per cent. As would be

expected on anatomic grounds, the majority of such findings occurred in cases with involvement of the cerebral hemispheres. Although recorded in too few cases to be of great significance, the relative occurrence of macular sparing as compared with macular splitting should be mentioned. There is certainly no indication in the table to support the classic view that macular splitting is more apt to occur with involvement of the optic tract. On the other hand, the finding of sparing of the macula exclusively in the tumors of the hemispheres may have some relative value. It will be noted that the majority of such cases are recorded for locations where the optic radiations could be involved. Therefore it would be logical to assume that the fixation reflex pathways, the corticotectal tracts[2] which lie just outside the radiations, would also be involved. This would lend support to the view of Verhoeff,[9] who believes that sparing of the macula may be the result of faulty fixation.

The finding of quadrantanopsia is not indicative of temporal lobe involvement in this series of cases. A study of the table appears to support the view of Walsh,[10] who states, "An homonymous quadrant defect in the visual fields suggests involvement of the dorsal or ventral band respectively in the optic radiation, and such an involvement may be in the temporal lobe." There was only 1 case in this series in which quadrantanopsia occurred in a proved temporal lobe tumor. On the other hand, there were 9 of the group which showed a homonymous hemianopsia.

Mention should be made of the specificity of the finding of bitemporal hemianopsia in localizing tumors to the chiasmal region. This can be verified from table III. It is important to note that not one single instance of bitemporal hemianopsia was recorded which could logically be due to dilatation of the third ventricle, although 54 patients with subtentorial tumors were examined by perimetric methods.

There were 56 cases in which either hemianopsia or quadrantanopsia occurred. Thus, field findings of localizing value were present in slightly over 25 per cent of the cases in which a perimetric examination was performed. The value of examining the fields of vision in any case suspected of harboring a brain tumor therefore should be evident.

The finding of anisocoria in a case of suspected brain tumor is said to have little localizing value, according to Walsh.[10] That inequality of the pupils with irritative cortical phenomena can occur has been borne out by Penfield and Erickson.[7] However, certain of the cases listed in the table could well have been unrecognized instances of Horner's syndrome. It will be noted that in the frontal, temporal and temporoparietal tumors, the larger pupil was most often to the side of the tumor, but the number of cases is too small to warrant any definite conclusions.

The small total number of conjugate palsies (5.6 per cent for the series) indicates how infrequently such a finding occurs. The greater number of upward gaze palsies in tumors of the pineal gland shows a distinct localizing value. Downward pressure exerted by a pinealoma affects the more superficial tectal layers of the midbrain early. It has been shown experimentally that the corticotectal fibers which reach the roof of the midbrain are those having to do with vertical movement.[4] It is anatomically difficult to explain the upward gaze palsies noted for the 2 cases of frontal lobe tumor presented in table V.

Nystagmus is said to be present in a great majority of cases with cerebellar tumor.[10] This statement is substantiated by the present series, where 20 of 58 cases showed this finding, an incidence

of 34.5 per cent. As stated earlier in this paper, the lack of uniform classification in the records studied makes further conclusions valueless except to reiterate that the motor connections for ocular movement were probably involved in most of the other cases. The total of 39 cases in which nystagmus was recorded gives an incidence of 10.9 per cent.

The much greater incidence of abducens nerve involvement in the cases studied confirms the fact that the sixth cranial nerve is relatively less protected in its long intracranial course. An increase in intracranial pressure alone may be enough to implicate this nerve by shifting the position of the brain stem. There is little localizing value from abducens palsy, as may be seen in table VI. A review of the records shows further that the lateralization of the palsy has no significant bearing on determining the side of the tumor. The lesser number of oculomotor and trochlear palsies would indicate either a shorter intracranial course or a more protected position. It will be noted that such ocular palsies occurred in only 13.4 per cent of the total series. The convergence palsies noted in the table are difficult to accept unless one assumes that these may have been unrecognized cases of internuclear ophthalmoplegia.

SUMMARY AND CONCLUSIONS

In a series of 358 cases of intracranial tumor the following ocular findings were present:

1. The incidence of papilledema was 59.5 per cent. Tumors which were above the tentorium presented papilledema in 53.3 per cent of the cases, while the subtentorial group had a 75.2 per cent incidence. Almost all the cases were bilateral.

2. Optic atrophy was found in 12 per cent. The cases with primary optic atrophy were mainly those in which direct involvement of the nerve, chiasm or tract was possible. Those with secondary atrophy occurred where the tumor could produce an early rise in intracranial pressure.

3. Abnormal findings were present in slightly less than half the cases where the visual fields were examined. The findings were of localizing type in slightly more than 25 per cent of the cases tested.

4. The statement of Walsh[10] that pupillary changes are of little localizing value was supported by the present study. Anisocoria occurred in 12 per cent of the series.

5. Defects of conjugate movement occurred in 5.6 per cent of the cases. Involvement of upward conjugate gaze in pineal tumors appeared to have the greater value for localization.

6. Nystagmus was present in 10.9 per cent of the series. It was a frequent finding in cases of tumor of the cerebellum.

7. Extraocular muscle palsies were found in 13.4 per cent of the cases. The incidence of abducens, oculomotor and trochlear involvement reflects their anatomic arrangement. Such palsies were of little value in lateralization.

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DISCUSSION

DONALD J. LYLE, M. D., Cincinnati, Ohio: Many conditions affect a statistical study of this nature, especially in reference to symptomatology which is influenced by three chief factors:

1. Stage of involvement
2. Rapidity of involvement
3. Nature of involvement
   a. By direct pressure of invasion
   b. By remote production of general bulk
      x. Through tumor development
      y. Through production of obstructive hydrocephalus

Rapidly growing tumors are usually associated with tumefaction, swelling, or edema, at first intracellular, then extracellular, and because of the rapidity of growth there is less opportunity for compensatory adjustment to the added bulk, both in the neighborhood of the tumor and in remote locations. Local or focal symptoms of intracranial tumors may be confusing and misleading. The oculomotor, trochlear, trigeminal and facial nerves may be stretched by a shifting of the brain and confuse the location of the lesion by their implication. For these reasons, one must pay particular attention to the first symptoms as later ones may be misleading in their localizing and diagnosing value. One must remember that localizing signs and symptoms may be produced not only by tumors but by vascular lesions of various types, injuries and other conditions. Therefore, the presence of certain symptoms or syndromes, although they may be of localizing value, is not necessarily diagnostic of the type of lesion causing them.

I believe that the differences between the percentage found by Dr. Petrohelos and Dr. Henderson and those of other investigators lie in the fact that their material as a whole, as they state, was subjected to earlier diagnosis and treatment. This applies to all of the symptoms they enumerate and describe in which there has been a discrepancy in statistical figures.

As to the difference in the amount of papilledema between the two sides, I am in agreement that there is very little. Perhaps in tumors of the middle fossa there may be some dissimilarity of elevation at the disc, but in these cases one may be dealing with direct pressure on the ipsilateral side in the production of primary optic atrophy and indirect pressure on the contralateral side resulting in papilledema. These cases of Foster Kennedy syndrome or Gower-Patton-Kennedy syndrome were not found by the authors, probably because they appear usually in advanced conditions. I am quite sure, however, that they are occasionally found in the average clinical practice. This syndrome is produced by tumors of the base of the frontal lobe, tumors of the middle cranial fossa which include hypophyseal adenoma and anterior temporal lobe tumors as well as the meningiomas, craniopharyngiomas and less common neoplasms. In these cases there is, of course, a difference in elevation as well as in the pallor of the papilla.

It is unfortunate that the term used to describe the type of atrophy of the nerve fibers whose cell bodies are in the ganglion cell layer of the retina and whose axons pass out of the eye to form the optic nerve, chiasm and tract is frequently misleading and inaccurate. The term "secondary optic atrophy" simply means that destruction, both physiologic and anatomic, has followed an earlier involvement severe enough to kill the optic nerve, but it does not indicate what or where. If the destructive process occurs remote from the papilla, one might say from its appearance as seen with the ophthalmoscope that primary optic atrophy is present; if it appears at the papilla with the production of gliosis and fibrosis, a secondary optic atrophy is evident.

The essayists in this presentation wish secondary optic atrophy to mean atrophy following papilledema. I do not believe that I am helping to clarify the terminology to any great extent, but I would like to suggest the

use of the term "consecutive optic atrophy" to mean optic atrophy following papilledema. This term has been used for this type of optic atrophy by several neurologists.

There is very little definite information, but much has been said concerning the sparing and nonsparing of the macula. The authors' view is worth consideration.

Homonymous quadrantanopsia is possible if the lesion involves either the dorsal or ventral bands of radiation fibers. The difference between lesions posteriorly where the two bands of radiations course side by side, and in the forward temporal area, where they are separated by the temporal horn of the lateral ventricle and other structures, is that a posterior lesion frequently overflows into the adjoining quadrant fibers, whereas the anterior lesion shows a sharp horizontal line marking the lower boundary of the quandrantanopsia. (This is demonstrated in the charts and cases illustrating the condition.)

Although bitemporal hemianopsia resulting from dilatation of the optic recess in the anterior end of the third ventricle is an infrequent symptom compared with its production by tumors in the chiasmal region, it does occur. These conditions are not found in the authors' series probably because of the advanced stage, as mentioned above.

Pupillary changes have been noted both clinically and experimentally as arising from the stimulation of the (1) basal telencephalon, (2) midline thalamus, (3) subthalamus, and (4) midbrain, as well as from certain cortical areas. I agree with the authors that until further knowledge is forthcoming, anisocoria and indeed other pupillary abnormalities have merely a conjectural origin and location.

I might make a similar statement concerning nystagmus, both ocular and vestibular. Nystagmus has very little localizing significance save that lesions producing vestibular nystagmus of central origin occur usually in the posterior fossa. Infrequently, a temporal lobe tumor is found with the symptom of vestibular nystagmus, which leads me to the conjecture that there might be a vestibular cortical center in the temporal lobe adjacent to the auditory center.

Conjugate deviation of the eyes, usually irritative, sometimes paralytic (that is, paralysis of lateral gaze), may improve through a compensatory mechanism so that the symptoms which appear early, most frequently noted in acute brain conditions, may disappear. Conjugate deviations remain more permanent when produced by lesions in the brain stem.

If I were to suggest anything in addition to this comprehensive study, it might be to include the symptoms resulting from involvement of the trigeminal and facial nerves and their associated connections.

Tumors occurring in the middle fossa may produce pain from involvement of the trigeminal nerves or the gasserian ganglion which occurs in their confluence. The ganglion is joined with the pons by its sensory root, from which is given off ascending and descending fibers. In addition to the symptoms of pain, paresthesia and anesthesia with involvement of the trigeminal, trophic lesions are found along its course, especially with serious involvement of the cornea. Pain may also be found as the result of meningeal irritation as the dura and larger vessels are innervated by the trigeminal. Pain and trophic disturbances are therefore of localizing value in the presence of brain tumors.

The facial nerve is of great localizing value in intracranial tumors as its involvement points to different areas.

1. A difference between supranuclear and infranuclear lesions with their flaccidity and involvement of the eyelids is significant.
2. Infranuclear facial paralysis in association with abducens paralysis, the Millard-Gubler syndrome, points to a lesion in the brain stem in the vicinity of the lower pons.

I do not believe that exact percentages are authoritative, especially as they concern symptomatology. One must remember that he is working with living tissues exposed to various influences and because of the dissimilar factors at hand, they will not necessarily react in the same manner in every instance. However, in a closely studied series such as this, the statistics are of great interest and considerable importance. I wish to compliment the essayists on this concise and well documented study.

DR. HENDERSON: Dr. Petrohelos and I wish to thank Dr. Lyle for his discussion of our paper. It is obviously very difficult in a somewhat dry statistical presentation such as ours to include any particulars on individual cases. We hope in the future to be able to carry out the analysis more extensively. Thank you.

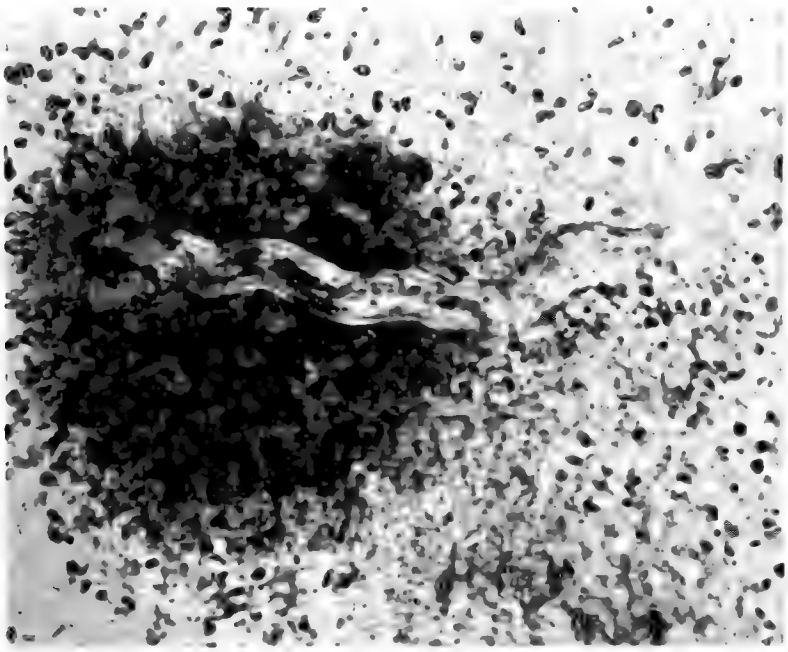


FIG. 1—Nematode larva in eosinophilic abscess in vitreous membrane. x400 AFIP Acc. 198761.

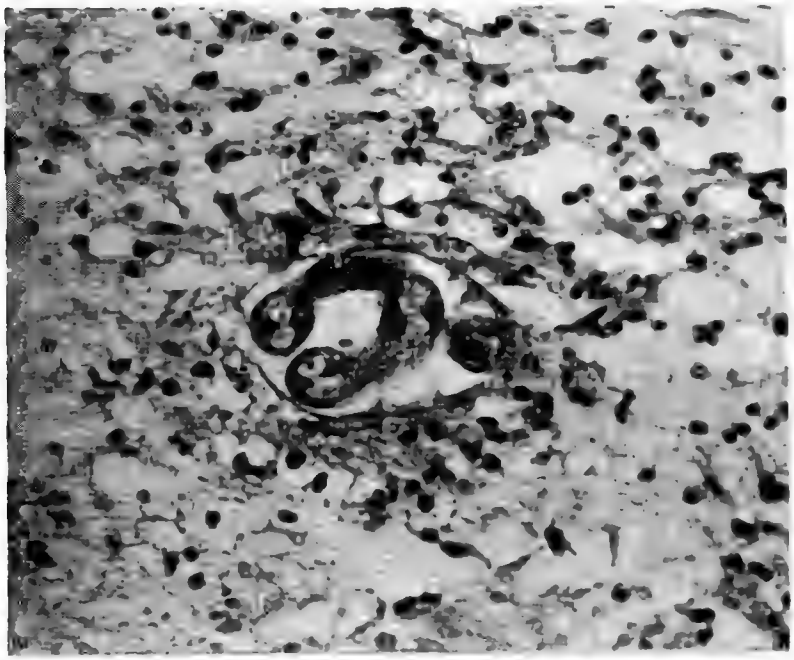


FIG. 2—Coiled nematode larva in vitreous membrane. x400 AFIP Acc. 293903.

NEMATODE ENDOPHTHALMITIS

HELENOR CAMPBELL WILDER
WASHINGTON, D. C.

ROUTINE examination of eyes at the Armed Forces Institute of Pathology disclosed a well-defined group in which the clinical histories and pathologic lesions were strikingly uniform. The eyes, with few exceptions, were from children. In most instances observation of a white pupillary reflex by the parents was the first evidence of ocular disease. On ophthalmoscopic examination there was seen behind the lens a pale mass with the blood vessels coursing over it, and a diagnosis of retinoblastoma led to enucleation.

Eosinophilic abscesses, sometimes surrounded by epithelioid and giant cells, presented a pathologic picture not accounted for by the more commonly recognized granulomatous lesions of the eye. Special stains failed to demonstrate bacteria, fungi, inclusion bodies, or organisms of any kind. As the lesions resembled those seen in helminth infections elsewhere in the body[2,3,14,15] and those described in the eyes of experimental animals,[19] it was decided to reexamine specimens exhibiting a suggestive inflammatory reaction in the hope of finding the responsible organism. Forty-six eyes, each from a different patient, were selected for this investigation. On the basis of previous microscopic examination, diagnoses of endophthalmitis, pseudoglioma, and Coats's disease, i.e., external exudative or hemorrhagic retinitis, had been made. Serial sections were prepared on all available material, which in no case was complete since the specimens had been sectioned previously. Notwithstanding this handicap, nematode larvae (figs. 1, 2, 3, 4, 5, 6) or their residual hyaline capsules (figs. 6, 7, 8)[2,14,15] were found in 24 eyes. In one case examination of over 2300 sections resulted in the discovery of a single larva (fig. 3), the entire worm being contained within 12 sections. In another eye, three larvae were found.

In 9 eyes the larvae were exceptionally well preserved. These were examined by Dr. B. G. Chitwood,[6] who gave the following report:

> So far as can be determined on the basis of the material at hand, the specimens are third stage hookworm larvae. No information as to species has been obtained. However, Ancylostoma sp., Necator sp. and Uncinaria sp. are possibilities. These specimens are the same stage but differ very slightly from Oesophagostomum larvae 24 hours after infection, as seen in sections of a pig's esophagus shown to us by Dr. D. A. Shark, Zoological Division, Bureau of Animal Industry. Furthermore, we are unable to distinguish between this specimen and a nematode seen in a pathologic section of a dog kidney.

The fact that the larvae in these cases have been recognized as hookworm does not rule out other nematodes (Strongyloides, Ascaris, etc.) as possible causative agents in endophthalmitis.

During the course of this study an unusual coincidence led for a time to the incrimination of a nematode larva which had nothing to do with the ocular lesion. In those eyes which were sectioned in paraffin, filariform larvae, very different in appearance from the

From the Armed Forces Institute of Pathology.
Preliminary report presented at the Congress of the Pan-American Association of Ophthalmology, Miami Beach, 1950.
Presented at the Fifty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 8-13, 1950, Chicago, Ill.

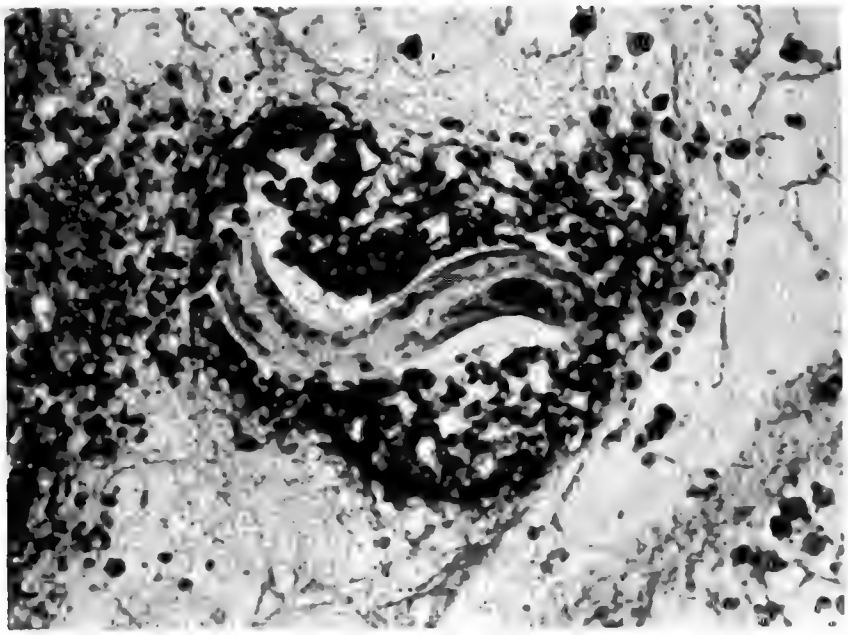


FIG. 3—Nematode larva surrounded by eosinophils in vitreous chamber. x220 AFIP Acc. 298563.

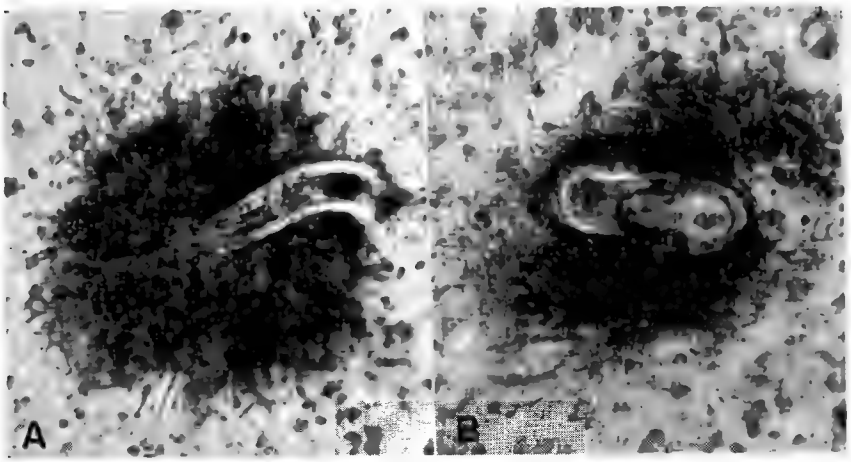


FIG. 4—Nematode larva in eosinophilic abscess in vitreous chamber. Serial sections showed the body partly extended and the tail coiled. A. x350 AFIP Acc. 69623; B. x400 AFIP Acc. 69623.

larvae within the abscesses, were found apparently within the globe. They were all complete, unsectioned larvae and were not surrounded by cellular exudate. As in a fourth case the larva lay on top of the section, it was decided without question that they were contaminants, probably deposited by a parasitized fly on the slides while they were drying on the warm plate.

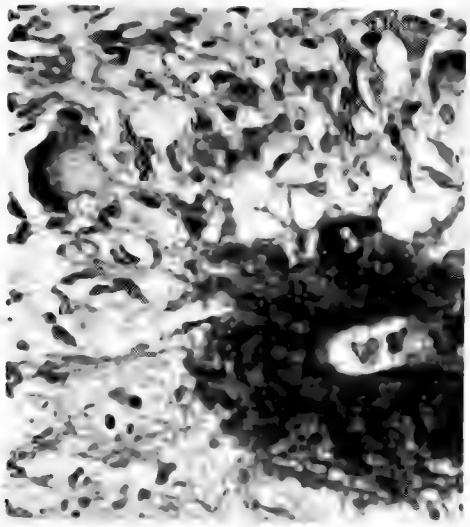


FIG. 5—Degenerated worm surrounded by necrotic eosinophils, epithelioid cells, and giant cells. x400 AFIP Acc. 202593.

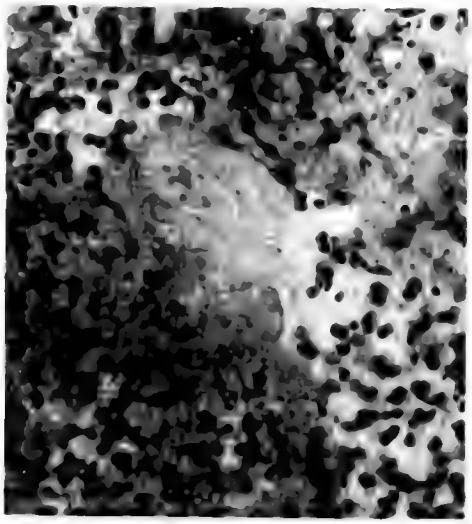


FIG. 6—Nematode larva, tangential section, surrounded by hyaline material. x450 AFIP Acc. 285189.

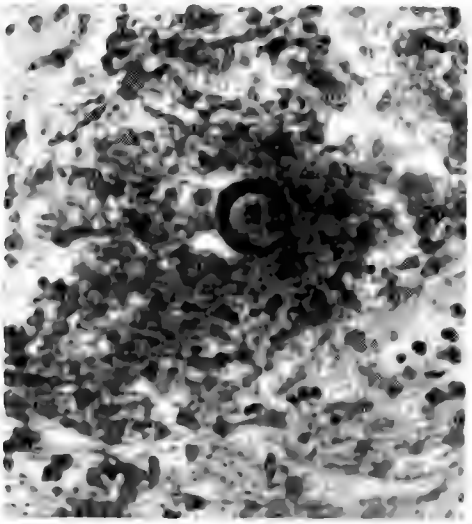


FIG. 7—Cross section of hyaline capsule surrounding completely degenerated worm. x400 AFIP Acc. 69623.

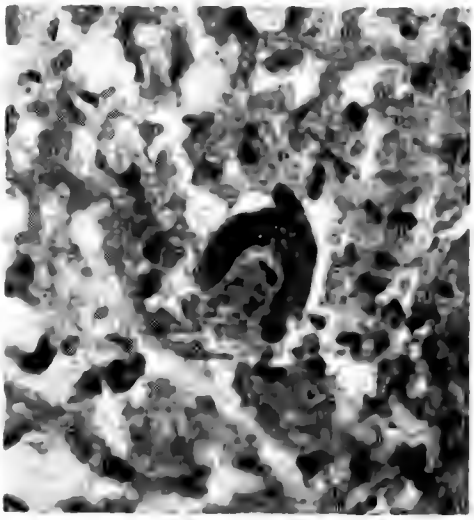
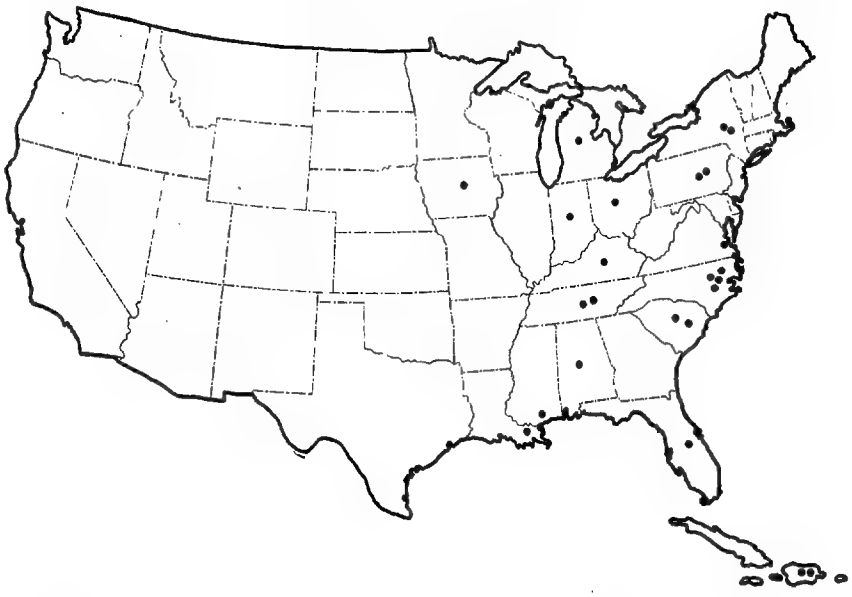


FIG. 8—Hyaline capsular fragment in older lesion. x604 AFIP Acc. 79868.

CLINICAL DATA

The 24 eyes in which the presence of larvae was established were all from children. The youngest was 3 years of age, the oldest 13, and the majority were of preschool and early school age: 3 through 5 years, 12 patients; 6 through 9 years, 9; 10 through 13 years, 2. Fourteen of the eyes were from girls, 10 from boys. Twenty-one of the patients were white and 3 colored. The specimens included 16 right eyes, 6 left, and 2 with side unspecified. In 2 instances lesions in the remaining eye indicated bilateral involvement. Although the majority of the patients were from

GEOGRAPHIC DISTRIBUTION



the southeastern United States, they were by no means limited to this region (see map). It must be taken into consideration, however, that the children from northern and western states may have lived in or visited other localities. A preoperative diagnosis of retinoblastoma had been made in 20 instances, pseudoglioma in 3, and in 1 in which the inflammatory reaction was particularly fulminating the clinical diagnosis was panophthalmitis. Anderson[1] observed that glioma (retinoblastoma) was the usual clinical diagnosis in ophthalmomyiasis when the larvae were located subretinally. Neither helminth infection nor any systemic disease was mentioned in the records of 20 patients. One had a history of old nematode infection. One child was cachectic, one had frontal headaches at the time of onset of ocular symptoms, and another, continued ocular pain and visual loss following meningitis eight years before enucleation. Usually there were no clinical signs of local inflammation. In addition to the 24 proved cases, there were 22 which were believed, on the basis of a similar pathologic picture, to be probable nematode endophthalmitis although larvae were not found. In general the clinical pattern and geographic distribution closely paralleled those of the proved cases, although 3 patients were adults and 1 was from as far west as the state of Washington.

HISTOPATHOLOGIC OBSERVATIONS

On microscopic examination, an inflammatory membrane was generally seen in the funnel of the detached retina, but it rarely involved the ciliary body (figs. 9, 10). In most, although not all, instances the anterior segment was comparatively free from evidences of inflammation. Cataract, however, was an occasional complication, and sometimes the posterior capsule was perforated, possibly by the larvae. Retinal, subretinal and vitreous hemorrhages were often present. Serous exudate usually occupied the subretinal space.

The most characteristic lesion was the eosinophilic abscess with a center in

FIG. 9—Site of larva in retinal fold indicated by arrow. Delicate vitreous membrane. Subretinal serous exudate. Retinal detachment. No involvement of anterior segment. x4 AFIP Acc. 201971.

FIG. 10—Site of larva in vitreous membrane indicated by arrow. Subretinal serous exudate. Retinal detachment. No involvement of anterior segment. x4 AFIP Acc. 198761.

which the cytoplasmic granules tended to become basophilic as a result of necrosis (fig. 11). Surrounding the abscesses were epithelioid cells, occasionally with giant cells (fig. 5), and inflammatory granulation tissue infiltrated by eosinophils, lymphocytes, and plasma cells which frequently were multinucleated, some having as many as five or six nuclei. There was considerable variation in the relative number of these cells in different lesions, apparently depending on duration and on the stage of disintegration of the larvae. Polymorphonuclear leukocytes were not conspicuous except in very early cases.

The eosinophilic abscesses were distributed on the underside of the retina, in retinal folds (fig. 9) and in the vitreous membrane (fig. 10). They also marked the sites of entrance of the larvae from the choroidal vessels, involving the inner layers of the choroid and breaking through Bruch's membrane (fig. 12). In many eyes they were found only on serial sectioning. Although it was in the abscesses that the larvae were located, every abscess did not contain a worm. This corresponds to the experience of O'Connor and Hulse,[15] who in a study of the generalized lesions of filariasis observed the organisms in all stages of degeneration in abscesses, and who concluded that failure to find them on serial sectioning was an indication that the filariae which had provoked the characteristic reaction had completely disintegrated. Intraocular lesions were not included in their investigation. In eyes studied for the present report the older lesions had undergone fibrosis but retained their tubercle-like pattern, and many of them still contained fragments of hyaline capsule (fig. 13). In case of long standing inflammation, granulation tissue and hemorrhage were replaced by dense fibrous membranes. Scars, which undoubtedly marked the tracks of the larvae, passed from the inner layers of the choroid and through the retina, interrupting Bruch's membrane and often forming chorioretinal adhesions (fig. 14). In a few cases calcium deposits were seen in the lens and retinal folds and a little bone formation in the choroid, features which might well add to the difficulty of x-ray differentiation from retinoblastoma. Cholesterin slits (fig. 15), masses of mononuclear cells, and even "ghost cells" beneath the retina had sometimes led to a diagnosis of Coats's disease.

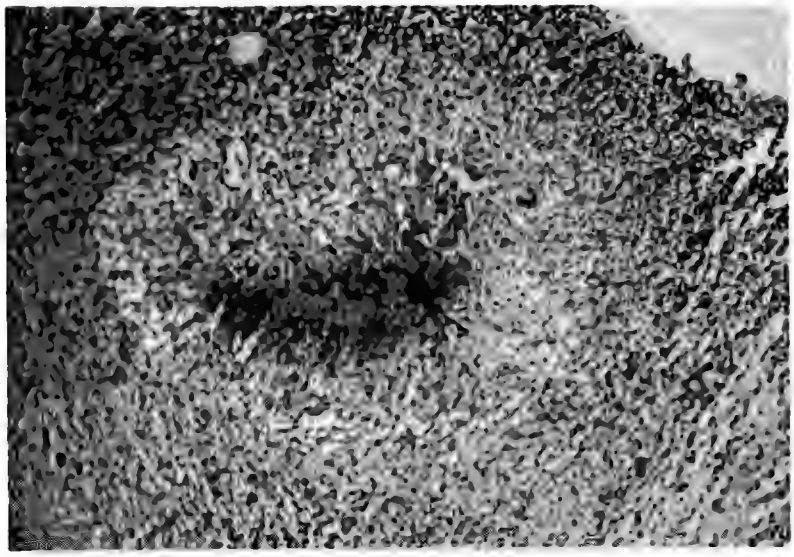


FIG. 11—Eosinophilic abscess surrounded by epithelioid cells and by inflammatory granulation tissue. Note hyaline fragment. x125 AFIP Acc. 79868.

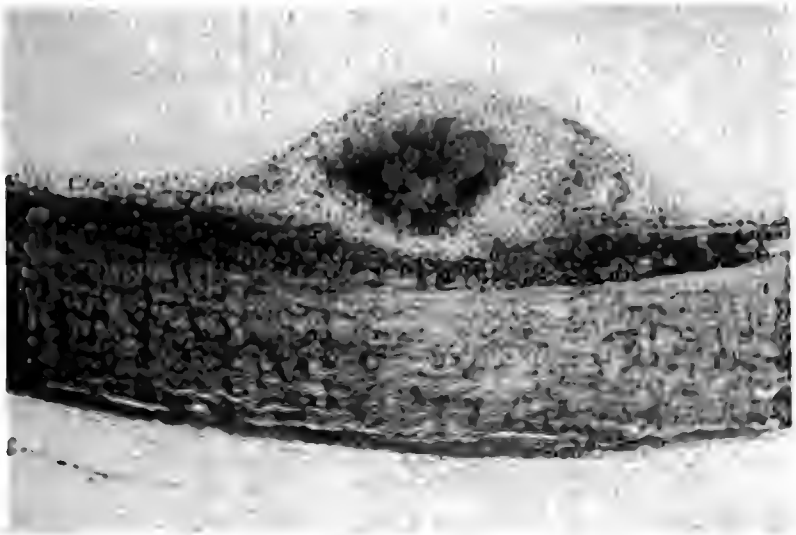


FIG. 12—Eosinophilic abscess destroying the inner layer of the choroid and interrupting Bruch's membrane. x48 AFIP Acc. 132401.

DISCUSSION

The chance occurrence of nematode larvae within the eye in nematodiasis is well known. There are excellent reviews in the literature of the reported cases of intraocular nematodes,[4,8,13] most of which have been filaria, particularly Onchocerca volvulus[7] and, less often, Wuchereria bancrofti and Filaria loa. Generally these parasites have been observed clinically in the anterior chamber, and nearly all the patients were

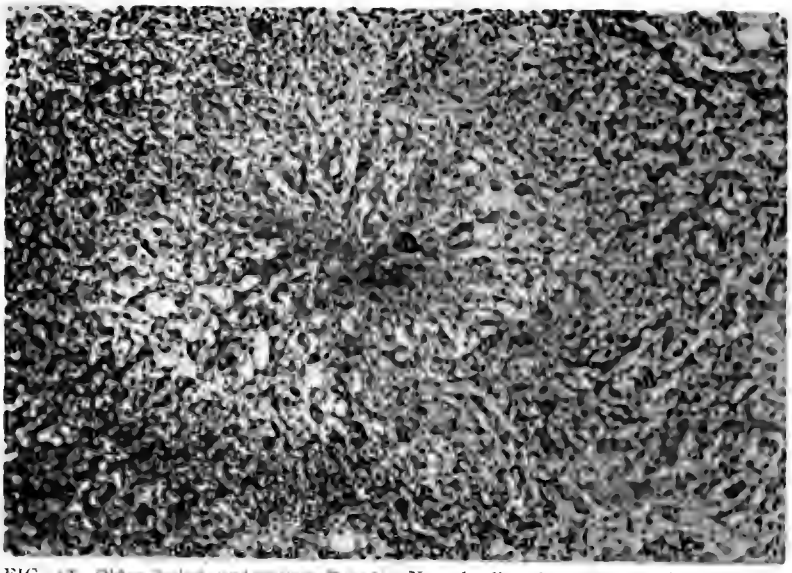


FIG. 13—Older lesion undergoing fibrosis. Note hyaline fragment. x125 AFIP Acc. 79868.

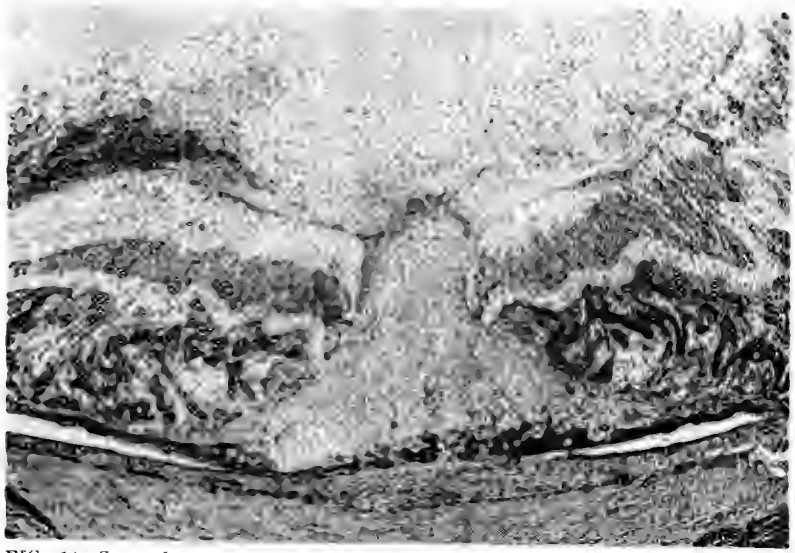


FIG. 14—Scar of entrance of the larva from the choroid, through the retina into the vitreous. x48 AFIP Acc. 293903.

from tropical regions.[16,17] Four reported cases are of particular interest either because of type of worm or geographic distribution. Sen and Ghose[18] removed a worm identified as Gnathostoma spinigerum from the surface of the iris in the eye of a Siamese. Although there had been recurrent iritis and hemorrhages in the retina and vitreous, recovery was uneventful except for the development of optic atrophy. Jones, Jordan and Sullivan[13] reported the case

of a 42 year old white woman who lived on the West Coast, the last 5 years in Portland. She had never been in the tropics. An adult type of round worm measuring 9 mm. and thought to be W. bancrofti was observed in the anterior chamber but unfortunately was lost on removal so that positive identification was impossible. Hosford, Stewart and Sugarman[12] removed 6 worms of Thelazia californiensis from within the eye of a 35 year old man. No further trouble

FIG. 15.—Subretinal mass of inflammatory tissue and organized hemorrhage with cholesterin slits in this case of nematode endophthalmitis (see figures 4 and 7) led to a diagnosis of Coats's disease. x125 AFIP Acc. 69623.

was experienced. Calhoun[4] observed a living Ascaris larva in the eye of an 8 year old Georgia boy. The larva increased in size during a period of five weeks, after which it died and was completely absorbed. Iridocyclitis and secondary glaucoma, which were present in the early stages of the infection, subsided.

In nematode endophthalmitis as seen in the cases from the Institute the location of the lesion indicates blood-borne infection. The larval parasites gain entry to the host by one of two possible routes — through the skin or through the mouth.[3,9] If they enter through the skin, they reach the right heart by way of the venous circulation whence they are carried through the lung to the left heart and so through the carotid, ophthalmic and ciliary arteries into the eye. If they enter by the mouth, they are probably transported directly to the intestine. They may then penetrate the intestinal wall and be carried by the venous circulation to the right heart and thence reach the eye by the same route as if they had entered through the skin. In either case migration through the lung, demonstrated by Füllerborn[10] and others, readily accounts for their presence in the eye. Choroidal lesions indicate that the metastases are generally to the choroid, as in neoplasms, rather than to the retina, and that the larvae reach the retina and vitreous by direct

invasion. Further proof of this was the case of Heath[11] in which a larva was seen leaving a choroidal vessel by perforating its wall. Cataract associated with hookworm disease has been accredited to anemia, toxemia, or a combination of the two by Calhoun.[5] He did not observe intraocular larvae clinically, and the eyes did not come to microscopic examination. He regarded retinal hemorrhages as the result of toxins, whereas Fülleborn[10] thought it possible that they resulted from larval emboli.

SUMMARY

Forty-six cases which had been diagnosed pathologically as pseudoglioma, Coats's disease, and endophthalmitis, and which showed similar inflammatory reactions, were the subject of special study. With few exceptions the 46 patients were children, the greatest number from the southeastern United States. In most cases a clinical diagnosis of retinoblastoma had preceded enucleation. Nematode larvae or their residual hyaline capsules were found in 24 eyes. In 22 others the characteristic reaction justified a tentative diagnosis of nematode endophthalmitis. In no instance was a parasite found in the original routine sections. Serial sections were necessary to demonstrate the larvae in every case. In 9 eyes the larvae were exceptionally well preserved and were identified by Dr. B. G. Chitwood as those of hookworm. The exact species remains to be identified.

CONCLUSION

The finding of intraocular larvae by serial sectioning and the identification of the specific pathologic reaction that they evoke has led to the conclusion that nematodes play an important and hitherto unrecognized role in blindness in children, and particularly in the production of pseudoglioma and Coats's disease in the United States of America.

ACKNOWLEDGMENT

Recognition is given to Dr. Georgiana Dvorak-Theobald, who, in 1947, on the basis of a lesion similar to those reported here, suggested the possibility of helminthiasis as a cause of intraocular eosinophilia; to Lt. Colonel Thomas Carlyle Jones, V.C., USA, who pointed out the resemblance of the ocular lesion to that commonly caused by helminths in other organs of animals; to Dr. Henry Rappaport, Armed Forces Institute of Pathology, whose experience in examining serial sections of an appendix riddled with lesions and finding only one nematode larva encouraged me in a seemingly endless search; to Dr. B. G. Chitwood, nematologist and Associate Professor of Biology, Catholic University, Washington, D. C., who contributed many hours to the identification of the parasite; to Mr. Lawrence P. Ambrogi, Chief of Laboratories, Armed Forces Institute of Pathology, and his staff, particularly Sgt. Evelyn F. Ballou and Mrs. Aileen Sevier, whose tireless efforts in preparing serial sections made this study possible.

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DISCUSSION

JOHN S. MCGAVIC, M. D., Bryn Mawr, Pa.: In bringing to our attention the fact that nematode infection can cause endophthalmitis, especially in children, Mrs. Wilder has done us a great service. I am sure you all realize the tedious effort required to examine serial sections of 46 eyes. Since over 50 per cent of the eyes selected for study because they showed a particular pattern of cellular reaction yielded positive evidence of parasites, this long search was well rewarded.

During what we would like to continue calling the last war I noticed that we could almost always find intestinal parasites in nonallergic soldiers with an eosinophilia of over 6 per cent. Although some of these soldiers also had choroiditis, the possible causal relationship did not occur to us.

During the past two years, I have observed a man who has had intestinal worms, eosinophilia of 11 per cent, and recurrent attacks of mild paramacular choroiditis with multiple retinal hemorrhages. While no other cause could be found, neither his internist nor I know how to prove or disprove a causal relationship. Mrs. Wilder's report makes it seem desirable to look for and eradicate intestinal parasites in such cases, as two of the 46 eyes were from adults.

We are especially interested these days in unraveling the mysteries of retrolental fibroplasia. In so doing, attention has been focused on other conditions which produce the pseudoglioma or leukocoria reflex. This endophthalmitis due to nematode infection explains some cases which we have been classifying as Coats's disease or as metastatic retinitis accompanying the exanthematous diseases of childhood. This is real progress.

Hookworm infection and malaria have been the two greatest obstacles to the economic development of the Southern States. Measures to combat them have been rather half-hearted. Perhaps the fear that children may lose eyes because they are infested with worms will stimulate a more vigorous fight against this common menace to the public health. If so, Mrs. Wilder's report will be more valuable than ever. I have a feeling that more parasitic diseases of the eye will be demonstrated in the near future.

It has been a pleasure to discuss Mrs. Wilder's excellent report.

THE CONTAMINATION OF OPERATIVE WOUNDS WITH COTTON FIBRILS AND TALC

L. R. DUSZYNSKI, M.D.

NEW YORK, N. Y.

BY INVITATION

GERMAN[2] emphasized that cotton or talc foreign bodies frequently escape detection in routine methods of histologic examination but can be easily demonstrated with polarized light. Routine microscopic sections of globes operated upon for glaucoma and subsequently enucleated were examined with a polaroid microscope.

Photomicrographs with polarized light show the location of the foreign bodies. Routine photomicrographs demonstrate the tissue reaction present in the foreign body field. The relationship between foreign body and tissue reaction can be compared by examining the same field with both technics (figs. 1 through 8).

Table I indicates the relative incidence of such material in routine sections of previously operated upon glaucomatous globes. While the number of specimens in each group is too small to permit statistical analysis, the low percentage in simple iridectomy would lead one to suspect that some factor separates it as an individual group. This factor is probably the minimal operative manipulation in this procedure, i.e., in the absence of bleeding, sponges are not introduced into the operative field and instrument technic limits contamination from glove talc.

Tissue reaction is dependent upon the site and nature of the foreign body.[4] The morphologic and optical properties of the material demonstrated in the polaroid photomicrographs is that of cotton or talc. The tissue reaction to each of these materials is fibroblastic and proliferative. Many observers have described this reaction in clinical and experimental studies.[1,3,5,11] The photomicrographs are confirmatory evidence in support of their previous findings and will not be elaborated upon at this time.

TABLE I

THE RELATIVE INCIDENCE OF FOREIGN BODY MATERIAL IN GLAUCOMA SURGERY

OPERATION	NO. OF SPECIMENS	NUMBER POSITIVE	PERCENTAGE
Cyclodialysis	37	31	84
Cyclodiathermy	5	5	100
Iridectomy	49	23	47
Iridencleisis	3	3	100
Iridotasis	1	0	0
Posterior sclerotomy	6	5	83
Sclerecto-iridectomy	9	6	67
Trepanation	106	83	78
TOTALS	216	137	73

The frequent presence of foreign bodies in eye surgery is significant in itself, and particularly so when one considers the care taken to prevent wound contamination with bacteria. "Physiologically inert" foreign bodies are not absorbed and do provoke tissue reactions. The low grade chronic inflammatory response, while apparently innocuous, is a hazard, actually and potentially. McCormick[5] reported a talc granuloma occurring fourteen years after muscle surgery.

Presented at the Fifty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 8-13, 1950, Chicago, Ill.

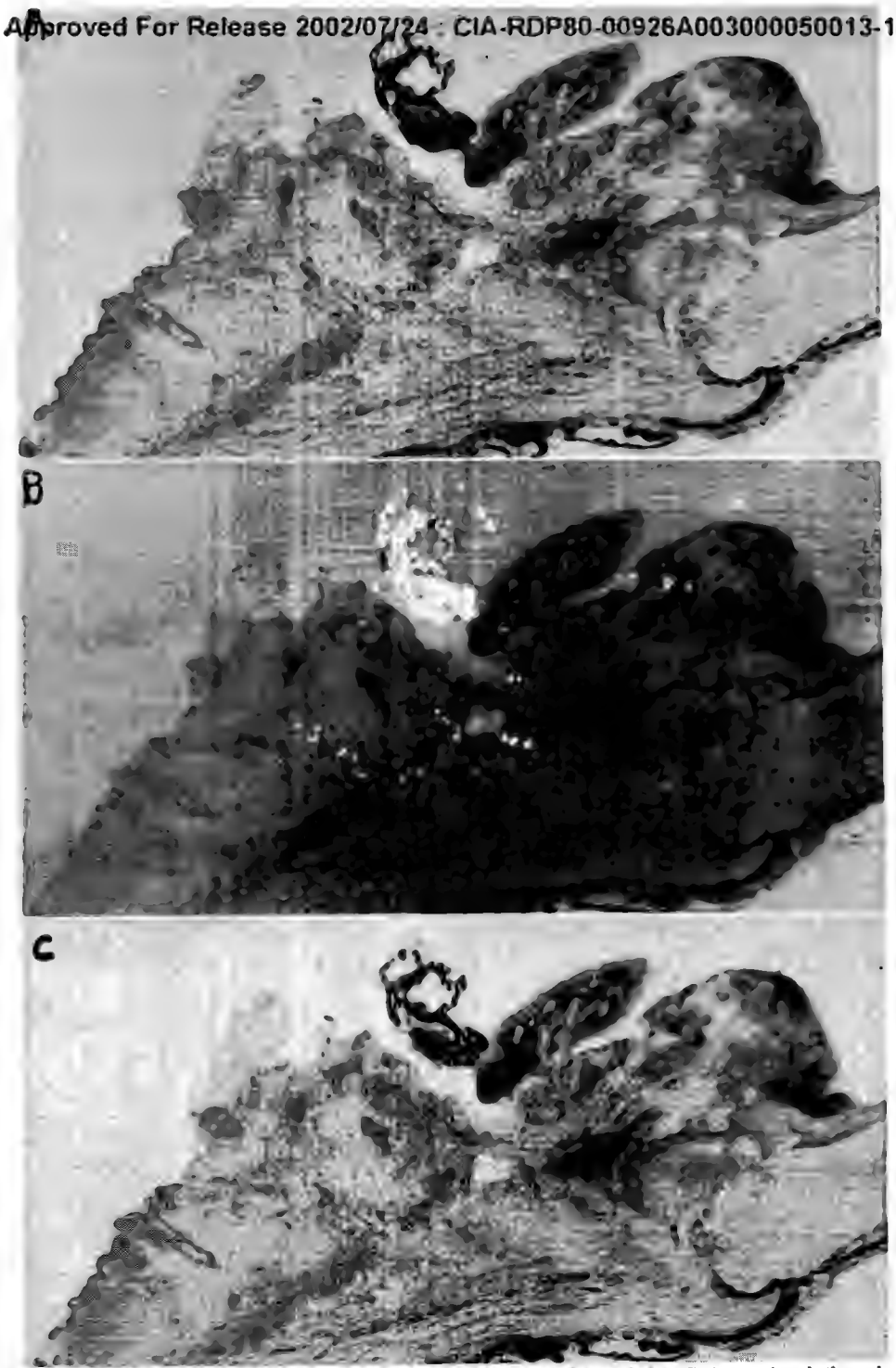

FIG. 1—Iridectomy. (A) Low power photomicrograph by routine technic. Globe sectioned through plane indicated by identification suture (black), and also passing through operative site. Note giant cells in tissue below identification suture. (B) Same field viewed by polarized light technic. Note distribution of foreign bodies (white) in the tissue. Compare location of foreign material with distribution of giant cells in (A). (C) Same field viewed by combined nonpolarized and polarized light technics. Note birefringent material in giant cells.

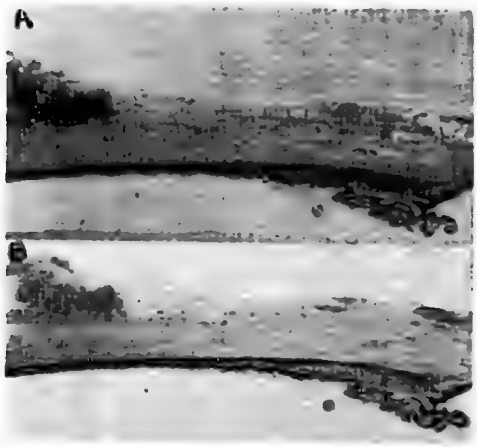

FIG. 2—Trepanation. (A) Photomicrograph by polarized light technic. Note birefringent material (white) posterior to opening. (B) Same field by routine technic. Note tissue response localized to foreign body material demonstrated in (A).

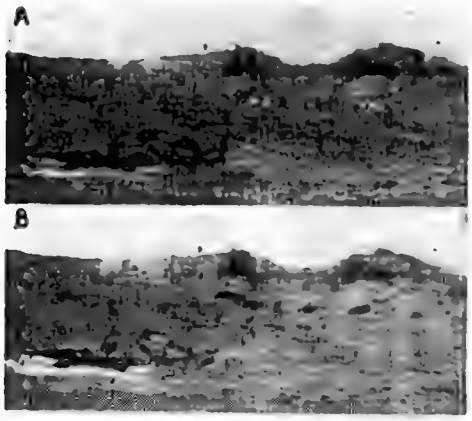

FIG. 3—Cyclodialysis and trepanation. (A) Photomicrograph by polarized light technic. Note birefringent material (white) in tissue above and posterior to operative site. (B) Same field by routine technic. Note tissue response and compare cellular reaction to location of foreign bodies demonstrated in (A).

SUMMARY

Routine sections of globes operated upon for glaucoma and subsequently enucleated were examined with a polaroid microscope. Photomicrographs demonstrate the presence of foreign bodies and the tissue reaction to them. The morphologic and optical properties of the foreign material are those of cotton or talc.

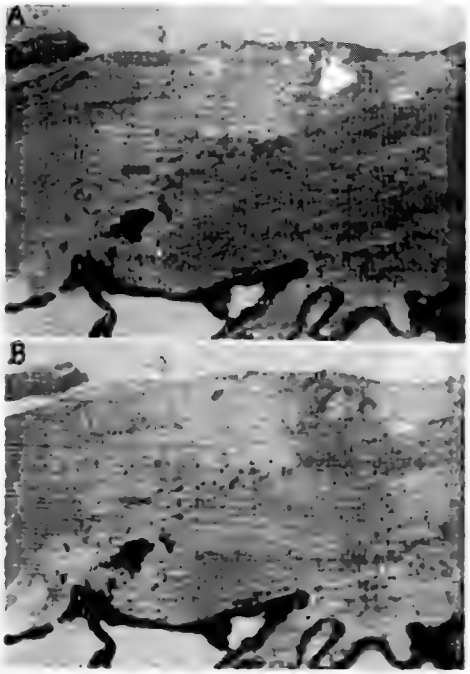

FIG. 4—Trepanation with iridectomy. (A) Photomicrograph by polarized light technic. Note many small foreign bodies (white) in tissue and large conglomerate foreign body located posteriorly in episcleral tissue. (B) Same field by routine technic. Note capsule about large episcleral foreign body demonstrated in (A).

The incidence of wound contamination by foreign bodies in glaucoma surgery is indicated and the possible significance thereof is mentioned.

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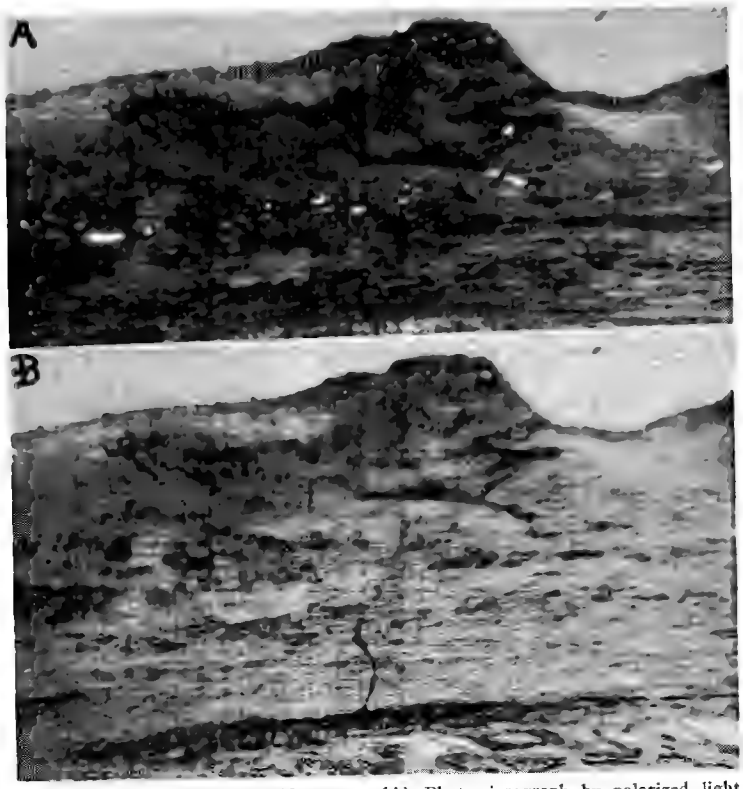

FIG. 5—Trepanation with iridectomy. (A) Photomicrograph by polarized light technic. Note numerous foreign bodies (white) present in tissues. (B) Same field by routine technic. Note tissue reaction and compare cellular response in plane of foreign bodies demonstrated in (A).

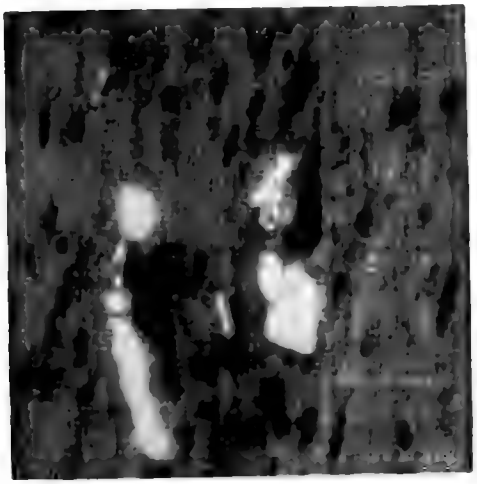

FIG. 6—High power photomicrograph of specimen in figure 5. Polarized light technic. Note foreign body material (white) in tissues.

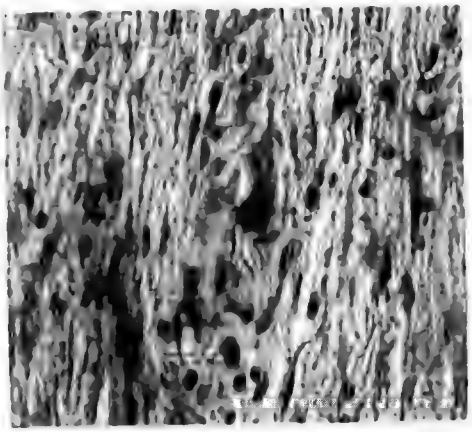

FIG. 7—Same field as in figure 6 by routine technic. One foreign body in focus. Note cellular reaction. The second foreign body lies in another focal plane and cannot be demonstrated simultaneously by this technic.

FIG. 8—High power photomicrograph of specimen in figure 2. Routine technic. Note cellular response in foreign body field.

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DISCUSSION

DERRICK VAIL, M.D., Chicago, Ill.: Like the ghost of Shakespeare's Hamlet, Dr. Duszynski has here unfolded a tale "whose lightest word would harrow up thy soul,
Freeze thy young blood,
Make thy two eyes, like stars, start from their spheres,
Thy knotted and combined locks to part,
And each particular hair to stand on end,
Like quills upon the fretful porpentine."

W. M. German showed in 1943 that bits of cotton and talc embedded in tissue can easily be demonstrated under the microscope by polarized light, standing out like "an electric sign at night." Dr. Duszynski has cleverly adapted this method to a routine study of eyes operated upon for glaucoma and subsequently removed. He reveals the astounding fact that 73 per cent of 216 such eyes contain cotton fibrils and talc and show unmistakedly the injury these foreign bodies produce. Without his method of study these harmful particles had escaped notice by even the most astute observers. His photomicrographs are more impressive and convincing than a thousand words. They sound the tocsin to alert us at once to this dangerous condition and stimulate us to take what measures we can to combat it.

At the last meeting of the American Ophthalmological Society, I introduced the subject of lint or cotton threads in the anterior chamber following intraocular surgery and showed

that clinically this condition probably was not rare and that these foreign bodies were capable of producing harmful reactions within the eye in spite of some reports in the literature to the contrary. I was alarmed by the enormous amount of lint from gowns, draperies, towels, and sponges that floated around in the air of the operating room. This lint dust, stirred up by the slightest move of anyone within the room, or entering or leaving it, swirled around and settled tenaciously on exposed instruments, solutions, sutures and the patient's open eye. In the beam of the strong operating lamp, the lint resembled white swarms of dancing midges. The saline for irrigation of the anterior chamber sucked into the glass syringe and held up to the light showed a nauseating quantity of minute pieces of lint, like thread worms, waiting to be injected into the anterior chamber.

The investigation of the problem showed that the pieces of free lint were capable of carrying harmful bacteria picked up in the air as it floated about. Ordinary measures to stop or reduce the amount of free lint particles were fruitless. In desperation the advice of Dr. Ralph M. Tovell of the Hartford (Conn.) General Hospital was sought. He advised that the laundry use a paraffin-base waterproofing material called Migasol P. J. (Ciba) upon all operating room linen not used to absorb moisture. The use of this material during the last four months at my hospital has apparently solved the problem of free lint. That there has been a striking decrease in the amount floating around is evidenced by the fact that at the end of a day's work in the operating room there is scarcely any lint seen on the mirrors of the overhead operating lamp, whereas hitherto a thick layer of it was obvious. I am, therefore, able to recommend to you without reservation this method for prevention of free lint. The method is simple. Migasol P. J. is added to the wash wheel after the laundry machine is loaded for the last time. The formula calls for one pint of Migasol P. J. for each 100 pounds of linen in the wash wheel. Linen used to absorb blood, serum and other body fluids should, of course, not be so treated.

Cotton sponges are another source of lint that can enter the anterior chamber. The matrix of the usual cotton sponge is very easily broken by rolling into the shape generally employed. Substitutes are used but these are not entirely satisfactory. Among them are cellulose and the compact "felt cotton" used so widely by neurosurgeons. I use the latter and find it satisfactory, although not perfect.

Powder from rubber gloves is another foreign body discussed by the essayist. Starch powder has replaced the older magnesium talc used on hands and rubber gloves, but particles of starch powder can easily enter the opened anterior chamber and can irritate the intraocular tissues. This danger can be alleviated by irrigating the newly gloved hands in a stream of sterile saline just before operating. However, those of us who do not wear gloves for intraocular surgery do not have this problem, as pointed out by Dr. Verhoeff in his discussion of my paper referred to. The assistants and the scrub nurse, however, do and they should be instructed to rinse thoroughly their gloved hands to wash off all traces of powder.

Instruments should be rinsed in sterile saline or dipped in zephiran and wiped with sterilized pieces of old fine linen just prior to use.

The fluid used for irrigation of the anterior chamber, should be contained in a closed sterile vessel and sucked into the syringe just before using.

Foreign materials of a nature other than lint are occasionally seen in the anterior chamber after surgery. These may be pieces of rubber (from rubber ball irrigators or Bell erisophake), bits of glass from a broken end of a glass tipped irrigator, pieces of rust and scale from plated instruments, oil drops from ophthalmic ointment, and bits of cilia. Methods to prevent the introduction of these substances, while fairly obvious, require more thought.

When we see an enlarged motion picture of a cataract extraction, for example, or see what we do when we wear a loupe during the operation, or when we examine with the slit lamp an eye newly operated upon, we are struck with horror over the damage we do to the tender tissues of the eyeball by our manipulations. This depressing situation is bad enough, although perhaps a necessary evil. At least let us take intelligent measures to obviate as far as possible the damage to an operated eye produced by things under our control. I shudder to think what we would see if we were to use polarized light in the clinical slit lamp examination of eyes which have been operated upon.

I am grateful to Dr. Duszynski for proving so convincingly in the laboratory what has been clinically observed, particularly for showing us how common this serious problem is, for alerting all of us to its danger, and for bringing up this important subject for thought and discussion that may result in far reaching benefits to our patients.

MALIGNANT LYMPHOMA OF THE UVEAL TRACT

EDMOND L. COOPER, M.D.
DETROIT, MICH.
and
JOHN L. RIKER, M.D.
ALPENA, MICH.
BY INVITATION

AN interesting diagnostic problem is presented by a case of malignant neoplasm with ocular manifestations. In the early stages of the disease the lesion was misinterpreted as inflammatory, and it was not until the enucleated eye was examined pathologically that the proper diagnosis was made. The disease from which the patient suffered was of a general systemic nature; yet for several months the only signs and symptoms were ocular, and for this reason the case should be of interest to ophthalmologists.

CASE REPORT

The patient was a 27 year old white man, who in November 1947 had begun to have pain and blurring of vision in the right eye. He consulted an ophthalmologist who instituted treatment for iritis. When after a month the iritis had not improved but continued to progress, he was referred to us. There had been no attack of pain, no redness or blurring of vision in either eye preceding the "inflammatory" condition for which he had been receiving treatment. The past history was negative except for two significant events. In 1944, while attending a radar school at Boston, the patient was hospitalized for an illness characterized by swelling of the lymphatic structures of the groin, diagnosed "lymphangitis." In the following year, when he was stationed at Okinawa, jaundice and severe headaches were the prominent symptoms in another illness for which he was sent to the hospital. During this time his weight, which on induction to the service had been 199 pounds, dropped to 180, and from then on he had experienced a very gradual but persistent loss of weight.

Presented as a *Clinicopathologic Case Report* at the Fifty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 8-13, 1950, Chicago, Ill.

When the patient was first seen, the left eye was normal and it remained so throughout the course of the illness. Vision with the right eye was 20/25. There was a mild ciliary flush. Slit lamp examination revealed numerous mutton-fat keratic precipitates on the corneal endothelium and cells in the aqueous. The iris was thickened but not nodular, and the blood vessels were engorged. The pupil was moderately dilated, and there were no posterior synechias. The fundus was easily seen. There was considerable edema of the optic nerve head and the surrounding retina. The retinal veins were moderately engorged and exhibited periphlebitic changes. Throughout the fundus, but particularly in the upper temporal quadrant, were numerous retinal hemorrhages. The vitreous was free of hemorrhage. The intraocular pressure was 50 mm. Hg (Schiotz).

The diagnosis was neuroretinitis, retinal periphlebitis and retinal hemorrhage (Eales's disease to be ruled out), iridocyclitis and secondary glaucoma. The patient was admitted to Harper Hospital where paracentesis controlled the glaucoma. The inflammatory condition of the eye continued unchecked in spite of energetic treatment during the ten days the patient remained in the hospital. The iris became more thickened, and it was impossible to keep the pupil dilated. Posterior synechias developed. Large nodules, 3 or 4 mm. in diameter, began to form in the iris, and the anterior chamber became shallower as the thickening of the iris increased. In general, the appearance of the anterior segment suggested a chronic granulomatous process, and it was thought that the etiologic factor might prove to be tuberculosis, brucellosis, Boeck's sarcoid, or one of the other chronic granulomas.

While the patient was in the hospital, a thorough survey failed to provide a clue to the etiology. No foci of infection were found. Roentgenograms of the chest were normal. Skin tuberculin and brucellergin tests, the Frei test, blood agglutination tests for tularemia and brucellosis, and smears of the prostatic

secretion gave no positive information. Kahn test, urinalysis, and routine blood counts were normal. The differential leukocyte count was repeated twice and neither time was it unusual. The only abnormal finding in the physical examination was moderate bilateral inguinal lymphadenopathy with no tenderness. Biopsy of an inguinal lymph node was done, and the pathologist's report was "chronic lymphadenitis of undetermined character—no evidence of sarcoidosis." In short, nothing was found to which the iridocyclitis or neuroretinitis could be attributed.

Following the patient's discharge from the hospital the iridocyclitis progressed. The iris nodules became larger for a time and then gradually grew smaller. Dense posterior synechias and a pupillary membrane formed, so that the fundus could not be examined. In February 1948 vision was reduced to perception of hand movements. The intraocular pressure was subnormal. A course of streptomycin therapy was given without any apparent effect on the disease.

In March, because the eye was hopelessly blind and was rapidly becoming a cosmetic defect, and also because it seemed impossible to establish the correct diagnosis without pathologic study, the eye was enucleated. No difficulty was experienced except that dissection of the conjunctiva and isolation of the rectus muscle tendons were hampered by considerable thickening and brawny induration of the conjunctiva, episcleral tissue, and Tenon's capsule. The patient's total leukocyte count during this hospital stay did not exceed 9,500, and the differential count was normal.

During the following six months the patient's condition gradually declined. In May, a painless swelling of the right testicle suddenly appeared. It was relatively soft and uniformly smooth with no nodularity, and attained the size of a peach. This was the first clinical indication (unless the inguinal lymphadenopathy can be considered) of generalized disease. The testicle was removed and studied pathologically. At this time the differential blood count was normal. The liver and spleen were palpable and there were no indications of lymphadenopathy. Later the same month a cervical lymph node was removed and studied.

The treatment of the eye in the early stages of the disease included all accepted measures, both local and general, for severe uveitis. It was not until June 1948, when the pathologic diagnosis of neoplasm in the eye had been further confirmed by examination of the testicular tumor, that deep roentgen therapy was begun. Irradiation was directed not only to the orbit and the testicle but also to all of the lymph node areas which might constitute potential foci of metastasis, and to the pelvis, abdomen and mediastinum. Supervoltage roentgen rays were employed over all of the areas except the two axillas, where deep roentgen therapy was used.

In July 1948, the total leukocyte count was 4,500 and the differential count was normal. In the terminal stages of the illness the other testicle became involved, and there was clear-cut clinical and radiologic evidence of massive involvement of all the deep lymphatic structures of the abdomen, thorax, neck, and head. The patient died in October 1948. Autopsy was not permitted.

PATHOLOGIC OBSERVATIONS*

The enucleated eye was sent to the Armed Forces Institute of Pathology, and a report received in April 1948 gave the first indication that the lesion was malignant. The conjunctiva, episclera, uveal tract, retinal pigment epithelium, sensory retina and optic nerve were infiltrated with closely packed cells (figs. 1 and 2). These sometimes resembled lymphocytes, having small round, deeply staining nuclei and scanty cytoplasm

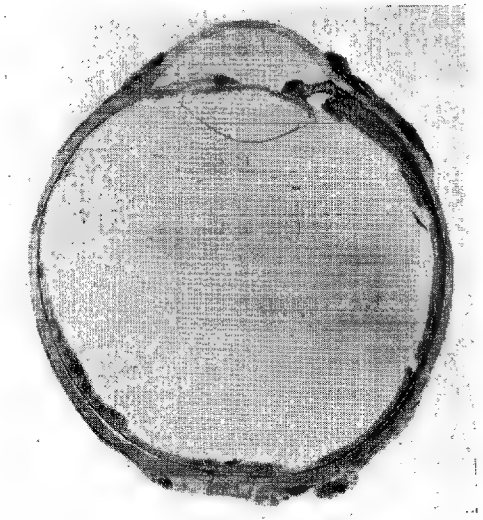

FIG. 1—Diffuse infiltration of the uveal tract, retina, and epibulbar tissues by tumor cells. x4. AFIP Acc. 205805.

*Pathologic reports furnished by Helenor Campbell Wilder, Registry of Ophthalmic Pathology, Armed Forces Institute of Pathology, Washington, D. C.

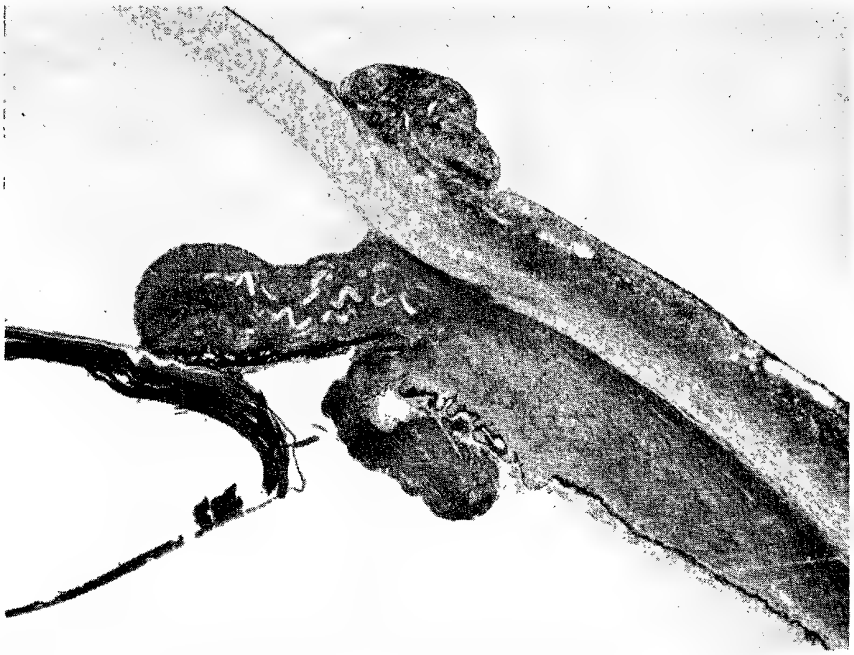

FIG. 2—Dense neoplastic infiltration with thickening of the conjunctiva, iris, and ciliary body. x16. AFIP Acc. 205805.

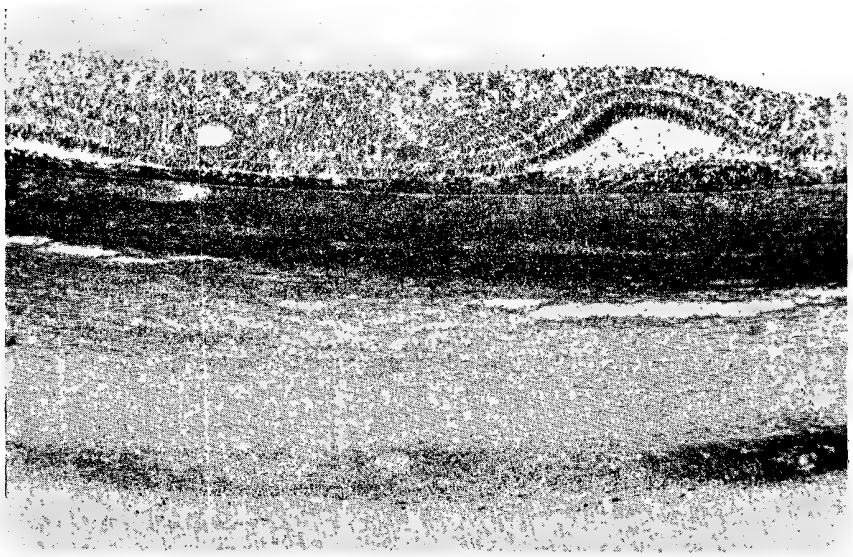

FIG. 3—Tumor cells with large nucleolated nuclei in the ciliary body. x705. AFIP Acc. 205805.

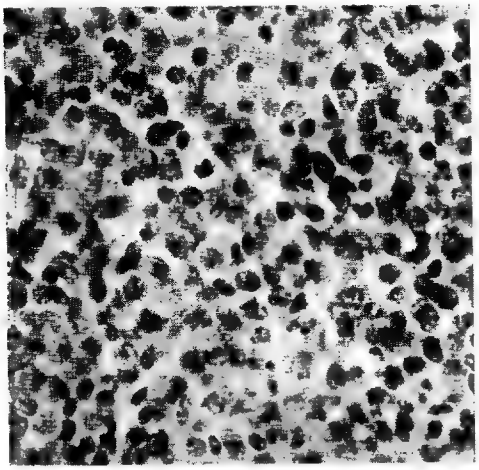

FIG. 4—Reticulum fibers surrounding tumor cells in the ciliary body (Wilder reticulum stain). x435 AFIP Acc. 205805.

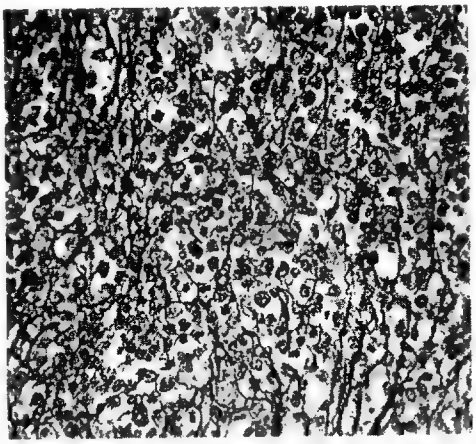

FIG. 5—Infiltration of the choroid and retina by tumor cells with partial destruction of retinal architecture. x48. AFIP Acc. 205805.

which was not always discernible. More often, however, the cells were larger, with rather abundant, ill-defined cytoplasm and large, oval or round, vesicular, nucleolated nuclei (fig. 3). These large cells approximated in appearance the cells of the germinal center of lymph follicles. A reticulum stain demonstrated a close association between the reticulum fibers and tumor cells in many areas (fig. 4). There were anterior and posterior synechias and a pupillary membrane. The lens showed subcapsular degeneration and wrinkling of the capsule. The retina was separated and folded in several places, and there was almost total loss of normal retinal architecture (fig. 5). The diagnosis was lymphoid tumor, probably metastatic reticulum cell sarcoma.

The lesion of the inguinal node which was removed when the patient was first hospitalized for treatment of the eye was diagnosed by the Harper Hospital pathologist as chronic lymphadenitis (fig. 6). Following the identification of the ocular neoplasm, a representative slide of this lesion was forwarded to the Armed Forces Institute of Pathology. The diagnosis of lymphadenitis was concurred in, although it was regarded as possible that the process represented an inflammatory reaction to adjacent neoplasm.

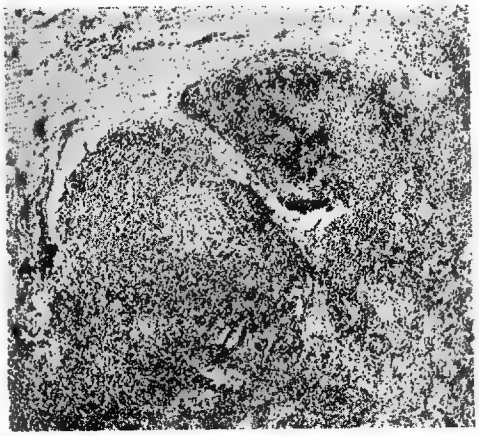

FIG. 6—Reactive inguinal lymph node. x76. AFIP Acc. 205805.

A microscopic slide of the testicle was submitted to the Armed Forces Institute of Pathology. It also revealed malignant tumor composed of small cells with scanty cytoplasm and round, deeply staining nuclei (figs. 7 and 8). The tumor in the eye more nearly resembled

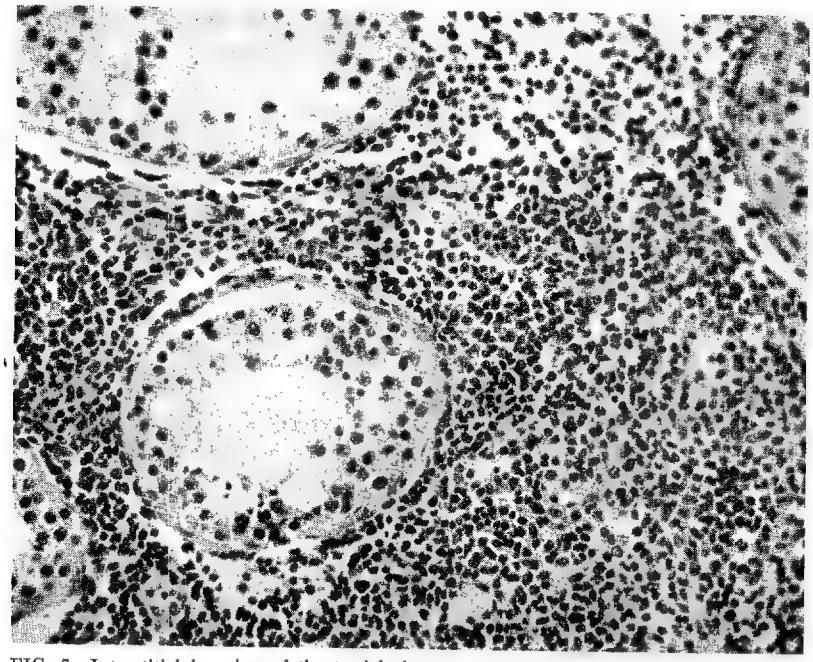

FIG. 7—Interstitial invasion of the testicle by tumor cells resembling lymphocytes. x435. AFIP Acc. 205805.

reticulum cell sarcoma, although that in the testicle had the appearance of lymphosarcoma. It is not always possible to make a definite differentiation between the two. Cells of reticulum cell sarcoma resemble the large cells in the germinal center of the lymph nodes, whereas a lymphosarcoma is composed of cells resembling the small cells of the node. Both have an origin from undifferentiated cells, and the variation results from the type of differentiation. The cervical lymph node, which was removed after the testicle, was studied elsewhere. The report stated that there was entire loss of normal architecture, the node being invaded by tumor cells similar to those in the eye.

COMMENT

Lymphoid tumors arising within the eyeball have seldom been described in the literature. In a report of 21 verified cases of lymphoid disease involving the eye and its adnexa, McGavic[3] found only one within the globe as against 20 which involved the lids, conjunctiva, or lacrimal gland. In Heath's series[2] of 67 ocular lymphomas, the globe was involved in only three. Sugarbaker and Craver,[4] reporting on 196 cases of lymphosarcoma, stated, "It is logical to suppose that lymphosarcoma is a disease which in the majority of cases begins and runs its early course in lymph nodes alone." In most instances of rapidly disseminating disease, it is impossible to determine whether a given organic involvement is secondary or primary.

Lymphoid tissues composed of follicles and sinuses do not exist in the eyeball or within the orbit; nevertheless, lymphoid tumors arising within the orbit are by no means unknown. They also occur in other locations where lymphoid

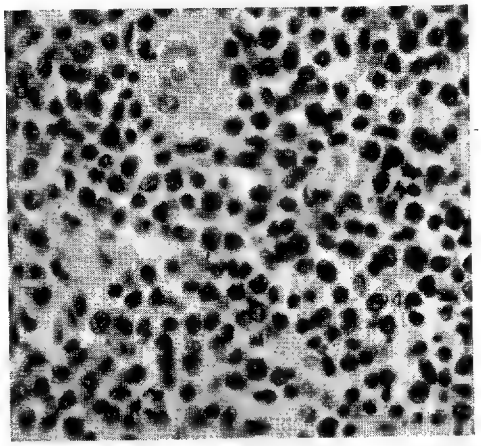

FIG. 8—Small tumor cells resembling lymphocytes in the testicle. x655. AFIP Acc. 205805.

tissues are not found normally, particularly in the skin where they are regarded as arising from undifferentiated cells around blood vessels. Possibly this tumor was primary in the eye, but the early episode of inguinal gland enlargement cannot be ignored. According to the pathologic reports from the Armed Forces Institute of Pathology, it more probably represented either metastasis from elsewhere in the body or one manifestation of a tumor arising in disseminated lymphatic tissue and from undifferentiated cells.

As is often the case, an exact classification of the tumor in this instance is impossible; it includes features of both reticulum cell sarcoma and lymphosarcoma. However, all malignant lymphomas, including Hodgkin's disease and the lymphoid leukemias, are considered manifestations of the same disease[1] and it is not unusual for them to exhibit a variable pattern in a single patient. The neoplastic process may be represented in one organ as giant follicular lymphoblastoma and in another as lymphosarcoma, or a tumor starting as Hodgkin's disease may terminate as lymphosarcoma, the transition being readily traceable in successive biopsies.

It is impossible to evaluate the causes which may incite lymphoid tissue to malignant growth. The history of our patient presents two episodes, one in 1944, the other in 1945. Although both were interpreted at the time as infectious, it cannot be proved or assumed that an inflammatory process preceded the neoplasm.

There is no specific hemogram in lymphosarcoma, and usually, when blood changes do occur, they are late in the disease and may be ascribed to radiation therapy. The only abnormality in the blood picture was moderately low hemoglobin late in the disease, and at no time did the patient have an abnormal leukocyte or differential count, although the last test was made only two weeks before his death.

It is agreed by most writers that the diagnosis of lymphosarcoma cannot be made clinically but must be established by biopsy. Early in the clinical course even biopsy may show what appears to be a reactive or benign proliferation of lymphoid tissues. Considering the important part played in inflammations by lymphocytes, it is sometimes difficult to evaluate their significance as part of an inflammatory or neoplastic process.

As far as treatment is concerned, all lymphomas are radiosensitive but not necessarily radio-curable. Obviously, the earlier treatment is begun, the better is the chance of cure, but the prognosis is usually poor.

SUMMARY

The malignant lymphoma involving the eye in the case presented was probably either metastatic or part of a widely disseminated lymphoid activity. The ocular process presented symptoms which simulated typical uveal inflammation, and the proper diagnosis was made only upon pathologic examination of the enucleated eye. In any case of severe uveitis with nodular iritis or re-

tinal hemorrhages for which no cause is found and which does not respond to treatment, it is important to consider the possibility of malignant lymphoid disease.

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A CONCEPT OF ALLERGY AS AUTONOMIC DYSFUNCTION SUGGESTED AS AN IMPROVED WORKING HYPOTHESIS

HENRY L. WILLIAMS, M.D.
ROCHESTER, MINN.

IT is important to remember that allergy has two aspects, the one clinical, the other immunologic. It is perhaps unfortunate that some allergists in turning from the one to the other aspect appear to insist that treatment of patients shall be carried on along exclusively immunologic lines although the diagnosis may have been made entirely on the basis of a practical clinical empiricism. The allergist in his role as immunologist has broadened our understanding of physiologic mechanisms that have to do with the organism's intolerance to interference from without. The immunologist in assuming the character of clinical allergist, however, often has seemed to be too rigidly insistent on the immunologic dogma that all the clinical manifestations that have become known as the allergies must have an antigen-antibody mechanism as a background of etiology and as a basis for treatment. Relatively recently the value of a clinical approach to many of the problems of allergy has received attention. Study of those factors that alter the threshold beyond which manifestation of allergy occurs, as Carryer[24] stated, has proved more fruitful in the care of many patients than has sole dependence on an antigen-antibody type of treatment. Nevertheless, most allergists insist on including an immunologic mechanism in the definition of allergy, although it seems probable that few of them insist on the demonstration of an antigen-antibody mechanism in making a clinical diagnosis of the condition.

I believe that this attempt to play the dual role of scientist and physician without separating the two parts sufficiently has caused some of the confusion in regard to the management of allergy. It seems to have been forgotten that the immunologic hypothesis was developed to explain certain syndromes whose confines had previously been laid out not by controlled experiment but on the basis of knowledge gained through observation and experience.

Von Pirquet's hypothesis of allergy[76] was based on the discovery of hypersensitivity in an immunized organism. He, however, considered these two terms to be mutually exclusive. It seemed impossible to him to consider as immune an organism which is protected against a disease, and at the same time is considered reciprocally hypersensitive to the same disease. This phenomenon could not be termed a paradox, he stated, because the word "paradox" should be applied only to an exceptional case, whereas the more one entered into this field of inquiry the more "according to lawness" the phenomenon was recognized to be. He, therefore, suggested a "new, general, nonprejudicial word" for the change of condition which the organism accomplishes, perhaps through the agency of an organic, living or lifeless *toxin*. According to von Pirquet[77] all that could be stated with certainty about a hypersensitive organism is that its readiness for reaction is altered. For this general concept of altered reactivity he suggested the ex-

From the Section on Otolaryngology and Rhinology, Mayo Clinic.

Presented at the Fifty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 8-13, 1950, Chicago, Ill.

pression, "allergie." Von Pirquet showed himself willing to alter details of the hypothesis as new observations were presented. Some of his followers, however, have not exhibited his intellectual adaptability.

Carryer stated that internists vitally interested in the problems of allergic disease are keenly aware of the shortcomings of a purely immunologic approach. He said, however, that inasmuch as the investigative thought over the past four decades has been greatly influenced by those whose background and experience were in the field of immunology, it was not difficult to understand why progress has been directed away from the nonimmunologic aspect of allergic disease. He recommended a critical review of the none too gratifying results of current, generally accepted and practiced tenets which have shortcomings of considerable magnitude.

DEFINITIONS AND DESCRIPTIONS OF ALLERGY

In considering the desirability of substituting a new and more flexible hypothesis for the antibody-antigen reaction, a review of some representative definitions and descriptions of allergy made by proponents of the immunologic hypotheses may prove useful. Rostenberg,[87] for instance, recently stated, "The best definition for allergy is the following one: 'Allergy is an acquired specific alteration in the capacity to react which is predicated on an antigen-antibody mechanism.'" In his opinion failure to include the antigen-antibody reaction has been the major reason for the confusion and lack of clarity which exist concerning the meaning of the word "allergy." He wished it to be clearly understood, however, that in the majority of allergic reactions an antigen-antibody mechanism is not demonstrable by conventional technics and in some cases not demonstrable by any known technics. This, he stated, does not mean that antibodies do not exist in these states; it merely means that we do not have as yet an appropriate indicator system to reveal their presence. He divided allergy arbitrarily into three immunologic patterns, the anaphylactic, the bacterial, and the eczematous type. He emphasized that it was important to realize that no clinical condition corresponds exactly to any one of the immunologic patterns listed. Many clinical conditions represent a combination of several of these types and, indeed, he considered that many varieties of allergic reaction do not fit into any of the three types given, but there is so little information about them that they cannot be set up as immunologic or allergic entities.

Rostenberg does not give a single instance in which he considered that treatment of cutaneous allergic disorders by immunologic methods was certainly beneficial.

Rostenberg then appears to declare that although factual data in regard to the presence or absence of an antigen-antibody reaction are absent in many of the conditions he considers allergic, conclusions arrived at by extrapolation and inference can be depended on to be correct and that such conclusions will serve to clear up the confusion and lack of clarity which exist concerning allergy. According to Bell,[14] however, extrapolation and inference have been the chief causes of the major errors in human thought.

On the other hand, Harley[44] stated that in his opinion the confusion and lack of clarity in regard to allergy were due to the tendency of allergists to invent new and perplexing terminology for phenomena which were fundamentally the same. In the present state of our knowledge it seemed to him impossible to give an exact definition to the term "allergy."

The "unitarian theory" which was developed to do away with some of the confusion of terms appears to have gained wide acceptance. As Zinsser, Enders and Fothergill[120] stated, this concept of allergy implies that the injection of an antigen into the tissues of an animal will lead to the development of a single antibody capable of producing the various manifestations of antigen-antibody union. This formulation has been further modified so that the various antibodies are considered to represent varied physicochemical changes in the same globulin.

Kahn[50] embraced the unitarian theory of allergy, but to the previous concepts of cellular immunity suggested by Metschnikoff,[67] and the humoral immunity suggested by von Pirquet,[76] he added the concept of the "third type of immunity" or tissue immunity. He rejected the suggestion that specialized cells would be entrusted with the entire defense of the body, stating that from the standpoint of phylogeny it would be reasonable to suppose that all cells possessed in some measure the ability to defend themselves against environmental stress whether that stress presented itself in the form of invading micro-organisms or some other form. He suggested that immune gamma globulin should be considered to be of two types, one of which is insoluble because it remains fixed in the cell, the other of which is soluble because it is dissolved in the blood serum and constitutes the circulating antibody. Kahn also considered that the differences between bacterial allergy and allergy in which circulating antibody can be identified were in degree rather than in kind. Allergy in his opinion is merely an instance of hyperimmunity and there are many examples of overaction of a normal physiologic mechanism.

In Bronfenbrenner's opinion,[16] the accumulated evidence indicates that whenever antigen-antibody reactions occur in vivo there is always a concomitant injury to the tissues of the host, as evidenced by both local and general symptoms. It appeared likely to him that the very mechanism which causes this injury to the host is also instrumental in bringing about such protective effects as the warding off of infection by immobilization of antigen by the antibody. Basically this injury consists of an inflammatory reaction which is evoked by the physicochemical changes in the environment and differs from ordinary inflammation only by its rapid onset and relatively stormy course. This inflammatory process is accompanied by both local and systemic symptoms resembling those elicited by the administration of histamine. Because of this resemblance and especially because of the highly suggestive experimental demonstration of the presence of histamine-like substances at the site of antigen-antibody union, he believed that the most widely held view today is that the inflammatory reaction is actually due to the liberation of histamine-like substances from antibody-laden tissues when they come in contact with the specific antigen.

Bronfenbrenner[16] also stated that phenomena called "immunity" and "anaphylaxis" are in fact merely different expressions of the same basic process of antigen-antibody union. Difference in the final outcome depends only on the extent and speed of this union and the consequent intensity of the injury to the host. Classical anaphylaxis as observed in animals and occasionally in man in his opinion is a laboratory artefact. Under natural conditions of exposure antigens enter tissues of allergic animals and of human beings much more slowly and in extremely small amounts; consequently, their union with antibodies is less explosive and the injury to the host is much milder than in

classical anaphylaxis. The symptoms elicited vary in form depending on the portal of entry, on the amount of antigen on one hand and on the relative local sensitivity of the involved tissues on the other. When so exposed, some individuals respond more easily to such immunogenic stimulus than others and these individual differences in reactivity seem to be determined by inheritance. The tendency to hyperreact is inherited, not the specific sensitivity itself.

A new hypothesis has been suggested by Wiener.[116] He stated that experimental work in relation to agglutinin and blocking antibody was the first definite demonstration of the incorrectness of the "unitarian hypothesis" of allergy. The evidence presented by his own work indicated that univalent and polyvalent antibodies are distinct entities as shown by the fact that they can be separated by natural means such as placental filtration. He pointed out that Witebsky and associates[118] partially separated these antibodies by dialysis in cellophane bags against distilled water. While the agglutinin proved to be associated principally with the resulting precipitate which contained most of the globulins, the blocking antibody or conglutinin remained mostly in the supernatant fluid together with the albumin.

Wiener offered the following criteria for allergy based on his theory:

1. The normal (nonallergic and nonimmune) state is that in which the body contains no induced antibodies specific for the antigen in question. Cognizance is taken, however, of so-called natural or normal antibodies.

2. The immune state is one in which the body has acquired large amounts of antibodies of the blocking type formed in response to the introduction of antigen into the body by either natural or artificial means. In this state an excess number of univalent antibodies are free in the plasma and other body fluids.

3. In the allergic state the body contains sensitizing (bivalent) antibodies attached to cells, with little or no free univalent antibodies in the body fluids.

4. Hyposensitization is the process of converting the allergic state into the immune state by repeated injections of antigen at sufficiently wide intervals to stimulate the production of potent blocking antibodies. This treatment is successful only when the subject achieves an adequate level of free univalent antibodies in his or her body fluids.

5. Desensitization consists in the injection of progressively increasing doses of specific antigen in rapid succession in order to saturate antibodies attached to body cells. This method, besides being dangerous, is often unsuccessful and the refractory state that ensues is only temporary due to the subsequent production by the body of additional bivalent antibody.

This hypothesis appears to combine Kahn's concept of tissue immunity with the concept of humoral immunity. Wiener pointed out a number of problems in immunology, previously unanswerable, that can be explained in the light of his new hypothesis.

While all these commentators differ considerably among themselves as to what constitutes allergy, they all insist that definition of the term "allergy" must contain a reference to an antigenic mechanism.

It is clear from a review of the literature of allergy, however, that the diagnosis of allergy in the clinic depends not on immunologic tests but on a well-taken history and careful observation of the patient.[4,26,35,47,68,79,104,108] It is obvious that Rostenberg, for instance, in classifying the various dermatologic allergies is depending on history and observation, else he would not so carefully point out that in the majority of such

patients immunologic evidence is unobtainable.

In discussing skin tests in diagnosis of allergy Tuft,[104] while insisting that they *are* of value in diagnosis when they can be obtained, pointed out that as in other laboratory tests the results must be correlated with the history and other clinical findings before an accurate diagnosis is possible. He found that unless a positive reaction can be obtained repeatedly, such a reaction cannot be considered specific but must be attributed to faulty technic, contaminated syringes, or irritating extracts. He divided all specific positive reactions into (1) nonclinical positive and (2) clinical positive. It seems clear that Tuft has made the diagnosis of allergy on the basis of clinical empiricism and merely hoped for confirmatory and possibly more specific information from skin tests.

Hansel[41] tacitly admitted that as a rule the diagnosis of allergy is made from clinical grounds alone when he stated that eosinophilia of the nasal secretions is the most dependable sign of allergy. The exact role of the eosinophil in allergy appears to be unknown. Petersen,[75] however, stated that he was able to produce showers of eosinophils by causing localized tissue anoxia. This finding suggests that eosinophilia can hardly be considered an indicator of an antigen-antibody reaction however valuable it may be as a diagnostic test for allergy.

Fox, Harned and Peluse[37] in discussing "borderline" allergy obviously were not considering allergy as occurring only among those patients who gave evidence of an antigen-antibody reaction.

Physical allergy. Not all allergists have insisted on an antigen-antibody hypothesis of allergy, however. Duke[33] stated that in his opinion in only a minority of patients with asthma, hay fever, urticaria, eczema and other manifestations of allergy can the source of the illness be traced definitely to contact with some material substance to which the patient is sensitive. He found that in the majority of patients, even after a most painstaking effort had been made through the use of history, physical examination, skin tests and clinical tests, the primary source of the disorder could not be shown to be sensitivity to substances such as pollen, dander, foods, drugs, or vapors. He was astonished to find, on the other hand, that a rather large proportion of patients with allergic conditions are sensitive specifically and solely to the action of a physical agent such as light, heat, cold, or mechanical irritation and indirectly to the action of emotional perturbation and fatigue. He termed such reactions "physical allergy."

There is a tendency among allergists, however, to deny that "physical allergy" as defined by Duke should be actually considered an allergy. For instance, Bronfenbrenner[17] stated that in his opinion it is unlikely that any real allergy (production of antibodies) to light exists. He remarked that although several authors have claimed to have succeeded in transferring light sensitivity passively, the lack of proper controls minimizes the significance of such findings.

In studying physical allergy Swineford[101] stated that in his opinion physical allergy usually is an associated allergic condition and not a primary reaction. It might be pointed out, however, that the evidence he presented indicates that the antigen-antibody reaction is secondary as much as it does that the physical reaction is secondary. Since this is the only paper I have found in the literature purporting to show that physical allergy in some manner is secondary to antigen-antibody allergy, and because there are several reports to the contrary,

it would appear that Swineford's findings need confirmation.

Peshkin[74] observed that 10 per cent of normal children reacted to skin tests with a variety of allergens. He also found that some of these "immunologically positive" children were later precipitated into acute episodes of clinical allergy by other presumably unrelated conditions, such as acute infections. He suggested that clinical reaction patterns, depending presumably on some other factor than the antigen-antibody mechanism, needed to be developed before clinical expression of allergy could take place. He termed such nonclinical sensitivity para-allergy.

Urbach and Gottlieb[106] suggested the term "pathergy" to cover both physical and antigen-antibody allergy. Physical allergy was to cover those cases in which an antigen-antibody mechanism could not be demonstrated.

It would appear that Peshkin and Urbach and Gottlieb were trying to escape from the horns of a dilemma posed by making diagnoses of allergy on a purely empirical basis, while at the same time insisting on the strictest adherence to the antigen-antibody concept of allergy in theory.

Selye,[95] however, reviewed the extensive literature that suggests that the organism could develop specific and nonspecific crossed resistance to various irritants without an antigen-antibody mechanism, and White[114] recently defined immunity to include resistance to physical stimuli in which an antigen-antibody mechanism apparently plays no part.

INADEQUACY OF THESE HYPOTHESES

Since it appears that allergists in general make the diagnosis of an allergic condition through history and physical examination, since the physical and immunologic allergies cannot be distinguished from one another by these means, and since the majority of clinical allergies, according to Duke, fall into the group of physical allergies for which clinically significant skin tests are not obtained, does not insistence on the antigen-antibody concept of allergy tend to lead physicians into the error of unnecessarily doing repeated skin tests and treatments with various antigenic substances in cases in which such tests and such treatment could not be expected to be of clinical benefit?

Does not adherence to the antigen-antibody hypothesis tend to concentrate therapeutic attention on attempts at hyposensitization alone, or at least to methods by which the manufacture of sensitizing antibodies can be diminished while increasing the production of blocking antibody or to methods which tend in some manner to influence antibody formation? Empirically it has been observed that attention to general nutritional factors, the use of vasodilators, trace minerals and various vitamins as well as psychotherapy have appeared to influence allergic manifestations in individual cases. In cases in which these measures exert a beneficial influence and in cases which are set apart as physical allergy is not the physician at a loss for a reasonable hypothesis on which to base treatment?

What can be the reason for this rather blind allegiance to the antigen-antibody hypothesis?

It seems to me that this attitude results from three factors. The first is respect to the memory of Clemens von Pirquet. The second is the impression that no adequate hypothesis for allergy is available to replace the antigen-antibody hypothesis. The third is based on the second and is the impression of many allergists that unless there are some stable reference points to restrain the diagnosis of allergy within reasonable bounds, the tendency to define allergy as a distaste for something or other, as in the lay expression "an allergy

to work," cannot be successfully combatted. This seems an entirely reasonable attitude; therefore, let us examine the second factor carefully.

It would seem from the increasing acceptance of the unitarian hypothesis of allergy and the work of Cannon and Pacheco,[21] Cannon and Sullivan[22] and Walsh, Sullivan and Cannon[111] that von Pirquet's hypothesis has outlived its usefulness. Thus a different hypothesis whose fundamental assumptions are not in conflict with the knowledge gained in the forty years that have passed since von Pirquet presented his paper seems to be required. According to Bell,[14] this is the normal orderly progress of the scientific method of thought.

It might be well, therefore, to examine the available data to see whether a foundation can be found on which a new formulation can be erected that avoids the difficulties of the old. It would appear that the suggestion that allergy may be a form of autonomic dysfunction furnishes such a foundation.

RELATION OF AUTONOMIC DYSFUNCTION TO ALLERGY

The Autonomic System and Functions

Cannon[20] observed that the sympathico-adrenal reaction was one of the mechanisms by which the body tended to restore physiologic equilibrium or homeostasis when it was subjected to stress in either its external or internal environment. Petersen[75] further extended the concept of the autonomic system. He stated that while the mechanisms designed to meet the environmental stress are manifold, the vast majority of these reactions are primarily autonomic. Phylogenetically, he considered that the various means of autonomic integration must have taken origin in the following order:

1. The primary method, when the organism was unicellular or consisted of a few cells, was chemical and enzymatic.

2. As the organism became more complex and a circulatory system developed, substances we call "hormones" were produced which circulate in the fluids of the body. Their prime purpose is to speed autonomic reaction.

3. The third method evolved when autonomic correlative efforts required not only speed in the processes of integration or restoration of physiologic equilibrium but direction of localization as well. For this purpose the anatomically defined autonomic nervous system, both sympathetic and parasympathetic, developed.

Petersen pointed out that these three components of the autonomic system are functionally inseparable; no disturbance can occur in any one element without immediately affecting the other two. He also noted that whatever autonomic alteration took place was immediately reflected in the behavior of the peripheral vascular bed, the arterioles, capillaries and venules. These reacted to environmental stress in a stereotyped manner, whether the stress arose in the external or the internal environment and whether it was due to physical agents, emotional perturbation, the invasion of micro-organisms or of nontoxic protein substances.

The existence of this stereotyped vascular reaction has received abundant confirmation.

By means of the method of Lombard,[61] observation of the functional reaction of the peripheral vascular bed to stress has enlightened us as to the fundamental functional changes resulting from the attempts of the organism to re-establish physiologic equilibrium or homeostasis. These were completely obscured as long as the pathologist restricted his observations to dead, fixed tissues.

Ricker and Regendanz,[83,84] for instance, found that a typical stereotyped

vascular reaction was present in inflammations of all types, and that an inflammatory reaction could not take place in tissue in which autonomic denervation had been done. They observed that in mild inflammation there was arteriolar and capillary dilatation with hyperemia. In somewhat more severe inflammation the arterioles were constricted with a slowing and clumping of the formed elements of the blood. In severe inflammation they found arteriolar spasm with dilatation of the contiguous capillary and venule. According to Oertel,[71] they gave convincing answers to criticisms of their findings.

Carrier[23] observed these same peripheral vascular reactions on exposure of the organism to differing degrees of cold.

Brown[18] observed this same type of arteriolar and capillary reaction in Raynaud's disease. He stated that this reaction represented a disruption of the normal co-ordination between arteriole and capillary. Krogh[57] has shown that the capillaries have an independent autonomic nerve supply and their caliber constantly changes. He also brought forward evidence indicating that the arteriomotor and capillomotor systems are able to respond in opposite directions to the same stimulus. Krogh noted that individuals exhibit a greater or lesser tendency to react to cold. He stated that although hyperreactivity may be a normal tendency in certain individuals, this tendency can reach an abnormal level. Brown also had found that in patients with the vasomotor neuroses the reactions to cold were similar to, but more marked than, the reactions of normal individuals and occurred at higher temperatures.

Mygind and Dederding[70] noted the same lack of co-ordination between arteriole and capillary in the skin of patients with Ménière's disease. They conclude that a similar reaction in the inner ear was the probable cause of the signs and symptoms of this disease. Lewis and Landis[59,60] observed the same arteriolar constriction with capillary and venular dilatation in acrocyanosis. Fremont-Smith and his co-workers[38] observed complete stasis in all visible capillaries during a chill; this stasis was due to constriction of the terminal arterioles. Parrisius[72,73] found lack of co-ordination among arteriole, capillary and venule in the skin of patients who had chronic simple glaucoma and Ménière's disease. Redisch and Pelzer[81] and Kennedy[53] found that while the premonitory symptoms of migraine might be due to vasospasm in larger vessels, the characteristic headache seemed to be due to this stereotyped lack of co-ordination between arteriole and capillary in the vasa vasorum of the involved extracranial vessels.

A most extensive investigation of the so-called vasomotor neuroses or dysfunctions has been made by Müller.[69] In many of these conditions including the allergic states, such as urticaria, vasomotor rhinitis, asthma, angioneurotic edema and the like, he found the same typical picture in the peripheral vascular bed. He termed the arteriolar constriction with capillary dilatation "the spastic atonic state," and noted, as other observers have done, that not all tissue areas were involved but that areas of arteriolar and capillary dysfunction would alternate with normal areas. He pointed out that the anoxia in the involved tissue would lead to increased capillary permeability.

Brown[18] observed destruction of the leukocytes in the involved capillary loops. Code[27] has shown that most of the histamine in the body of a human being is contained in the leukocytes. Destruction of the leukocytes in the capillary loop together with more or less injury to other involved cells would result in the release of histamine, which

also would increase capillary permeability. Depending on the type of cell injured, other toxic substances, such as heparin, or leukotaxine, the leukocyte promoting factor, necrosin and pyrexin, as observed by Menkin,[66] might be liberated. These substances could produce the same fundamental type of vascular change seen in all reactions of the organism to stress in the internal or external environment, including those found in the allergies. It has been pointed out repeatedly that allergy is primarily a vascular phenomenon. Rich and Follis,[82] for instance, found that the Arthus phenomenon could not be produced in an area devoid of blood vessels. Klinge[56] found evidence to indicate that in the pathogenesis of allergic lesions produced both clinically and in the laboratory, fibrous necrosis of the collagen fibrils is the earliest observable organic pathologic change. Similar changes in the ground substance also were observed by Rössle.[86]

The presence of this stereotyped reaction of the peripheral vascular bed has been observed in immune reactions in experimental animals. Bally[7,8,9] found constriction and spasm of the vessels of the rabbit's ear in histamine, peptone and anaphylactic shock. Szepsenwol and Witebsky[102] showed that three-day-old chick embryos contained the Forssman antigen and that the vessels constrict when Forssman's antiserum is applied directly. In microscopic observations on the behavior of the living blood vessels of the rabbit during anaphylaxis, Abell and Schenck[1] observed constriction of the arterioles. Wittich[119] observed the same reaction in the vessels of the chick embryo during active anaphylaxis. McMaster and Kruse,[64] on investigating the peripheral vascular reactions in anaphylaxis, found chiefly vasospasm and arrest of the circulation in sensitized mice. They found that sharply localized contractions appeared in many arteries. In those instances in which vascular spasm did not occur for a minute or two, the slowing of the circulation was the most prominent feature. Cells moving in clumps separated by plasma, as though they had become sticky and adherent, could be seen. This latter phenomenon, which they identified as sludging of the blood, was observed by Timonen and Zilliacus[103] in allergic reactions in the human being. It was their opinion that this did not result from an antigen-antibody reaction but depended on a more primitive resistance mechanism involving the reaction of the peripheral vascular bed.

The evidence appears convincing that a typical fixed, unchanging type of vascular reaction is present and is fundamental in that it is an indispensable part of the autonomic reaction to environmental stress. It also would appear reasonable to assume that in phylogenetic development the first stress to which the organism would need to adjust itself would be physical and chemical changes in the environment. It remains to be suggested, however, how, on the basis of this stereotyped reaction of the peripheral vascular bed consisting of arteriolar constriction with atonic dilatation of the capillary and venule, we can differentiate the allergies from other types of inflammatory reaction resulting from stresses occurring in either the internal or external environment. This differentiation appears to reside in the hypothesis of autonomic dysfunction.

Hypothesis of Autonomic Dysfunction

Eppinger and Hess[36] suggested and described the hypothesis of autonomic imbalance or dysfunction. This has been restated by Wenger[113] to bring it into line with more recent findings.

A. The differential chemical reactivity and the physiological antagonism of the adrenergic and cholinergic branches of the autonomic nervous system permit of a situation in which the action of one branch may predominate

over that of the other. This predominance, or autonomic imbalance, may be phasic or chronic, and may obtain for either the adrenergic or the cholinergic system. B. Autonomic imbalance, when measured in an unselected population, will be distributed continuously about a central tendency which shall be defined as autonomic balance.

The Role of Autonomic Dysfunction in Allergy

Kuntz[58] pointed out that some of the most characteristic manifestations of allergic disease are causally related to heightened parasympathetic or cholinergic activity. He was of the opinion that the so-called allergic state could not exist in the presence of a normal functional status of the autonomic nerves. It seemed to him that abnormal functional states of the autonomic nerves might be induced by the tissue reactions to the sensitizing agents in question, but on the other hand not infrequently the modified functional status of the autonomic system is a factor in the etiology of allergic disease. He stated that although many allergic manifestations undoubtedly result from the antigen-antibody reaction of the tissue elements, the manifestations of physical allergy cannot be explained on this basis. In either case, however, Kuntz noted that the functional disturbances bear essentially the same relationship to the autonomic nerves. These functional disturbances involve primarily tonic changes in the musculature of the visceral organs and especially in the vascular system. The cholinergic influence in allergic reactions of all types is indicated by the fact that regardless of which tissue is affected, adrenin affords relief. The general adrenergic reaction tends to counteract the effect of the local cholinergic stimulation wherever the disturbance may be.

Kennedy[53] also pointed out the importance of the autonomic system in allergic reactions. He observed that a sensitized person may exhibit allergic phenomena on emotion only when the autonomic system is "triggered" by such emotion and at the same time is in a reactive state. He felt that in time a system-habit reaction, referable to an unstable autonomic mechanism, might develop in such individuals. In migraine, he noticed the presence of arteriolar spasm with atony of the capillary and venule to be preponderantly on the same side of the body as the headache which he felt was on the basis of localized intracranial edema secondary to increased capillary permeability.

Belák[13] presented evidence which suggested that the production of immune substances took place secondarily to an autonomic reaction. This would suggest that the reaction of the peripheral vascular bed, which is an integral part of any autonomic reaction, probably preceded in phylogenetic development the appearance of an antigen-antibody reaction. In summarizing his own work and that of his associates, Belák classified immune substances in relation to the autonomic nerves as follows:

1. Sympathergic immune substances are the essential nonspecific antibodies, such as the alexins, opsonins and complement which are always present. He found their production to be increased by sympathetic stimulation and inhibited by parasympathetic stimulation.

2. Parasympathergic immune substances are the essential specific antibodies, such as antitoxin, precipitin, agglutinin and lysine. He found that the production of these substances is augmented by parasympathetic stimulation.

Kuntz[58] concluded that undoubtedly the specific immune substances are related to cholinergic nerves both of sympathetic and parasympathetic origin and that they respond to cholinergic (parasympathetic) stimulation according to a common mode.

Halphen and Maduro,[39] in studying spasmodic coryza, stated that when an

attack of vasomotor rhinitis is produced by contact with an allergen, the allergen is unable to produce symptoms unless the individual has a pre-existing functional disorder of the autonomic system. They considered that cold, humidity, physical factors such as sunlight on skin surfaces, and tobacco fumes certainly could produce vasomotor coryza solely by an autonomic mechanism. They were of the opinion that either local or general intolerance was fundamentally only the reflection of an acquired or hereditary autonomic dysfunction. That this is oversimplification of the problem is suggested by the circumscribed area of the lesions of allergy. White and Smithwick,[115] Kuntz and others have pointed out that the cholinergic fibers are the ones giving a localized discharge of stimuli.

In his hypothesis of the general adaptation syndrome Selye[95] suggested that environmental stress of all types called up a series of interrelated nonspecific systemic reactions of the body, the purpose of which is restoration of physiologic equilibrium. These reactions consist of physicochemical changes in the tissue fluids, reactions of the peripheral vascular bed and, especially, hormonal reactions. He pointed out that a review of the literature indicated the controlling position of hormones of the anterior pituitary and adrenal cortex in that such resistance reactions could not take place in the absence of the adrenal cortex or the anterior pituitary.

These reactions did not require an antigen-antibody mechanism for their completion. Selye was struck by the observation that these reactions were invariably the same no matter what stimulus called them forth. The "alarming stimuli" which Selye found capable of initiating the general adaptation mechanism are essentially the same as those previously described by Cannon[20] in his hypothesis of homeostasis, by Petersen[75] in his hypothesis of autonomic disintegration, and by Duke[33] in his hypothesis of physical allergy. Selye noted particularly that resistance could be developed to specific environmental stimuli without the mediation of an antigen-antibody reaction. He also pointed out the destruction of lymphoid tissue that occurred during the alarm phase of the general resistance mechanism. He called attention to the work of Sabin,[91] McMaster and Hudack,[63] Dougherty, Chase and White,[25,31] Harris and associates,[46] Ehrich and Harris[34] which indicated that the site of antibody formation may be the lymphocyte, or at least the reticulo-endothelial system. Rostenberg and Brunner[88] also critically reviewed the literature on antibody formation. They favored the hypothesis of enzymatic adaptation suggested by Burnet,[19] and stated that the somewhat divergent experimental findings might be welded together if it was considered that the primitive reticulum or undifferentiated mesenchymal cell might be the site of the enzymatic adaptation. Valentine, Craddock and Lawrence[107] have suggested that this work requires confirmation and that some of these conclusions may be erroneous. However, it can hardly be denied that this work does suggest that a stereotyped reaction of the autonomic system takes place before an antigen-antibody mechanism develops.

Selye divided the general adaptation mechanism into three stages: the alarm reaction, the stage of resistance, and the stage of exhaustion. The stage of the alarm reaction was subdivided into the stage of shock and the stage of countershock or reaction. The stage of shock bears much resemblance to the stage of exhaustion. It seems reasonable to assume, therefore, that a defect in the autonomic mechanism which calls out the stage of countershock, which eventually leads to the stage of resistance,

might well be considered a fundamental part of a dysfunction of the autonomic system. The similarity of the histologic picture of shock and allergy has long been noted.[97] For this reason the reacting cells have been termed the "shock organ." The great difference in the two conditions is the localized or focal part of the peripheral vascular bed involved in allergy as opposed to the generalized reaction in shock. That allergy might result from a disturbance of the anterior-pituitary, adrenal-cortical hormone also is suggested by the work of Kendall[51,52] on the physiology of the adrenal cortex.

In discussing the relation of the adrenal glands to immunity, White[114] stated that the factors which contribute to immunity are genetic, cellular, nutritional and hormonal. He stated that evidence is available that the adrenals play a significant role in the defense against physical, emotional and noxious stimuli. In his opinion elucidation of the role of the adrenal cortex has been obscured by studies with the hormone of the adrenal medulla since epinephrine has been established as a powerful stimulator of the rate of production of hormones of the adrenal cortex. While in this paper he emphasized the role of the adrenal cortex, White pointed out that the level of functioning of the adrenal cortex is markedly influenced by nutrition and by stimuli present in the environment. He favored broad use of the term "immunity" with a connotation of increased resistance to both nonantigenic and antigenic stimuli.

Tuft[105] recently stated that although the clinical findings of Duke have been verified repeatedly, no evidence has been presented as yet to indicate that the reactions depend on an antigen-antibody mechanism. Attempts at antibody demonstration have been generally unsuccessful. For these reasons it seemed likely to Tuft that the reactions of physical allergy are based on a physicochemical rather than an antigen-antibody mechanism. He also stated that there is no definite symptomatology or clinical picture characteristic of physical allergy by which it could be readily differentiated from antigen-antibody allergy.

It seems, therefore, that not only is "allergy" possible without the mediation of an antigen-antibody mechanism but that restriction of the term "immunity" by such an assumption of an antigen-antibody reaction may be incorrect.

COMMENT

The evidence presented seems to indicate definitely that the hypothesis of von Pirquet is no longer adequate as a working hypothesis because too many conditions recognized clinically as allergy fail to meet the criteria established by it. It is felt that a more adequate working hypothesis has been established by search of the available evidence. This evidence indicates that the peripheral vascular components of an autonomic reaction are the fundamental factors in the reactions by which the body attempts to restore physiologic equilibrium when subjected to environmental stress of any type. Allergy occurs when localized hyperactivity of this primitive immune reaction, with a cholinergic preponderance, develops. Consideration of allergy as a result of a dysfunctional preponderance of the cholinergic portion of the autonomic system seems to fit the available evidence better. Since the hormonal system is an integral part of the autonomic system as defined by Petersen,[75] along with the physicochemical reactions at the semipermeable membranes and in the tissue fluids, and the autonomic *nervous* system, the hormones of the anterior pituitary and of the adrenal cortex would be included.

There seems to be no particular reason for abandoning von Pirquet's term, "allergie," however. Its retention to

cover the conditions empirically diagnosed as allergies would serve to retain in our memory his great contribution to the study of immunologic mechanisms. An attempt to replace it with another word might add to the confusion produced by the tendency to give different names to identical physiologic processes, on which Harley[44] commented adversely.

Kahn[50] has defined an allergic individual (1) as one who may react maximally to stimuli that would produce only a mild autonomic response in a normal person, (2) as one who has a tendency to react to stimuli which would not develop a tendency to reaction in a normal person, and (3) as one who reacts to lesser stimuli than would provoke a similar reaction in a normal person.

Stiles and Johnston,[98] Baajol and associates,[6] Brown[18] and Müller[69] have presented evidence that this tendency to develop abnormal reaction to environmental stimuli is inherited.

Duke[33] has pointed out that a reflex type of allergic reaction may involve an area, a tissue or an organ. Petersen has spoken of "focal" autonomic dysfunctions.

A NEW WORKING HYPOTHESIS

Allergy may be defined, therefore, as an inherited predisposition to a localized type of autonomic dysfunction mediated by cholinergic fibers of the autonomic system. In these localized areas a stereotyped reaction of the peripheral vascular bed occurs consisting of arteriolar spasm with atonic dilatation of the capillaries and venules. This picture produced only by a maximal stimulus in a normal individual may occur in certain tissues and organs of an allergic individual in response to a normally minor stimulus. This reaction also may take place as a result of environmental exposure to stimuli to which a normal individual would not develop a reaction. The same degree of reaction may occur in an allergic individual in response to a much less severe stimulus than would be required to produce it in a normal individual. These reactions result in a greater or lesser degree of cellular damage and the release of histamine and other toxic substances depending on the type of cell injured.

The clinical picture of allergy may be produced by reaction of the peripheral vascular bed resulting in the production of anoxic capillary loops which may lead to typical allergic edema or necrosis and be classified empirically and clinically as allergy.

An antigen-antibody reaction may be associated with the vascular reaction and may aid in damaging the cell, but it is a secondary phenomenon, phylogenetically more recent than the vascular component of the autonomic reaction. This definition of allergy includes all types of allergy and yet serves to differentiate clearly allergic and nonallergic processes.

I have considered this focal type of autonomic dysfunction in a previous paper.[117] The fundamental assumption was that allergy is a clinical phenomenon. The diagnosis of allergy is primarily made empirically by observation of a gross lesion and of changes occurring in the function of organs and tissue in the light of past clinical experience. Observation may be extended secondarily by microscopic and immunologic methods. This is the manner in which the diagnosis of allergy has always been made, but it is inconsistent with a strict adherence to the hypothesis of von Pirquet.

Since von Pirquet and later Kahn (hyperimmunity) insisted that allergy is "altered reactivity," it should be possible to discover the normal prototype from which in each instance allergy has diverged. It would seem reasonable, therefore, to speak of three related but

not identical types of allergy: (1) physical allergy, (2) bacterial or tissue allergy, and (3) humoral allergy.

In physical allergy no antigen-antibody mechanism is present, cellular injury and the typical clinical picture being produced by anoxia. Its normal physiologic prototype can be considered the alarm reaction of Selye.

In bacterial or tissue allergy the fundamental autonomic (vascular) defense mechanism is retained, but it is suggested that in the process of phylogenetic development this defense mechanism has been supplemented by the development of protective antibodies. These protective antibodies are primarily attached to certain tissue cells but circulating antibodies may occur as a sort of by-product of cellular immunity, as suggested by Cannon. The normal prototype of tissue allergy could be considered to be granulomatous inflammation.

In humoral allergy, although antibodies are attached to cells, humoral or circulating antibodies are the outstanding feature. These may be the "blocking" univalent antibodies described by Cooke and associates[28] and Loveless.[62] It would appear that circulating antibodies take up some of the impact of the invading antigen so that less severe tissue injury is produced in the "host." The normal prototype of humoral allergy could be considered to be suppurative inflammation.

There is nothing in this concept of allergy to suggest that these three types of allergy are mutually exclusive. For instance, perennial vasomotor coryza which appears to be on the basis of a physical allergy may be frequently observed, especially in the cold months, and yet have seasonal exacerbations that appear to be on the basis of a specific sensitivity to pollen.

The working hypothesis of allergy as a type of autonomic dysfunction suggests the gradual growth in the animal organism of an increasingly more elaborate defense mechanism and that the new developments are added to the primitive stereotyped autonomic defense mechanism rather than replace it. The concept of allergy as primarily a hyperfunction or dysfunction of this stereotyped mechanism does not appear to be in conflict with any of the observed facts. It explains the gradual, rather than the abrupt, transition from one type to the other and why there may be a mingling of types. It also explains why, since the circulating antibody would appear to be a later phylogenetic development, it is not possible to discover evidence of circulating or sensitizing antibodies in so many patients with clinical allergies, nor to give them symptomatic relief by hyposensitization through the medium of the injection of specific antigens. Duke pointed out that in the majority of allergic individuals the reaction is not to a type of chemical stimulus (protein) that could reasonably be expected to result in antibody formation. At present there are several disorders such as Ménière's disease and myalgia in which the decision has not been made as to whether they are really allergic or not. With the present hypothesis of autonomic imbalance as a basis of allergy these disorders are readily classified as allergic, and successful treatment on a logical basis can be planned. This hypothesis throws the emphasis on the medical and psychosomatic aspect of the treatment of allergy which is receiving increasing consideration, as opposed to the strictly immunologic aspect.

THE MEDICAL TREATMENT OF ALLERGY

The advantage to be gained by consideration of allergy in the light of autonomic dysfunction is that all the allergies, whether an antigen-antibody reaction is present or not, can be treated from the viewpoint of clinical medicine.

Moreover, one type of treatment need not exclude others.

The effect of nonspecific stress such as inadequate nutrition, inadequate rest, inadequate amusement and relaxation in lowering the threshold of allergic reaction has long been known, but it has not been featured in allergic treatment because of a seeming reluctance to employ any but immunologic methods. The effects of physical agents in producing allergic reaction have been decried and there has been a tendency to deny them a place in allergy because immunologic treatment accomplished little.

Vasodilators. The vasodilators, however, have been known for a long time to produce favorable effects in physical allergy. The traditional form of attack on many of the allergies has been by vasodilatation. Many of these conditions were treated by the application of heat before any concept of allergy had been formulated. The treatment of vasomotor rhinitis and the vasomotor stage of the common cold by the hot mustard bath and by the opiates preceded the use of the antihistaminics by a considerable period.

Since the principal functional lesion in allergy appears to be vasospasm affecting the arteriole, an attempt to correct this dysfunction would appear to be the most logical approach.

The use of vasodilators for treatment of the allergies is based on the supposition that a vasodilator will release the spasm of the arteriole, resulting in renewed blood flow through the capillary loop which sweeps the contained cellular detritus and released toxic substances into the general circulation where they are immediately metabolized.

Duggan[32] has indicated that many of the vasomotor disorders affecting the eye, both those thought to be allergic and those not, are on the basis of arteriolar spasm. He has furnished a thorough review of the literature and added many suggestions in relation to the treatment of disease affecting the eye and its adnexa by vasodilatation.

Papaverine was one of the earliest vasodilators suggested for use in Ménière's disease by Müller.[69] Diehl[30] suggested its use in the common cold. Russek and Zohman[90] have found papaverine useful in the relief of cerebral angiospasm. Eppinger and Hess[36] in 1914 were among the first to suggest use of the vasodilator histamine for vasomotor neuroses such as angioneurotic edema. Müller recommended it for similar conditions in 1922, and Kling[55] in 1934 advised its use for rheumatic affections because of its effect as a vasodilator. Horton and his collaborators[48,49,96] have found histamine effective in the treatment of urticaria and Ménière's disease.

Weiss, Robb and Ellis[112] showed that histamine produced marked vasodilatation of the intracranial vessels of most individuals. These findings were confirmed by Wakim and his associates.[110]

The therapeutic effect of histamine on the allergies is the relief of vasospasm; its action is not essentially different from that of any other similarly acting vasodilator.

Harris and Moore[45] were the first to suggest the use of nicotinic acid for Ménière's disease. Bean and Spies[10] found that nicotinic acid and all of its pyridine compounds which contained the free nicotinic acid radical were vasodilators. Popkin[78] and Abramson, Katzenstein and Senior,[2] Crino and Lenzi[29] and Malaguzzi Valeri and Paterno[65] observed the effects of nicotinic acid to be similar to those of histamine. Roniacol (3-pyradine methanol), which is stated to be converted in the organism to nicotinic acid, is a long-acting vasodilator that can be given by mouth and has been found effective in the treatment of the allergies. Wakim and associates[109] found priscol (2-benzyl-4,5-

imidazoline hydrochloride) to be an effective vasodilator, and it has been used in the allergies with success.

Sympathomimetic Drugs. The fact that cholinergic nerves are concerned in the production of the allergies suggests that a beneficial effect should be obtainable by drugs which stimulate the adrenergic or paralyze the cholinergic system.

White and Smithwick classified the neurohormones and the most potent drugs which act on the sympathetic nerves as follows:

> Of the drugs acting on the sympathetic nerves, those which produce a stimulating effect are epinephrine or adrenalin, sympathin, ephedrine and amphetamine sulfate; those which produce a depressing effect are ergotoxine and nicotine. Of the drugs acting on the parasympathetic nerves, those which have a stimulating effect are acetylcholine and pilocarpine; those which are depressing are atropine and nicotine.

The advisability of prohibiting the use of tobacco in the allergies has long been debated. The studies of Roth[89] indicate that smoking promotes vasospasm. Use of tobacco in allergy, therefore, appears contraindicated.

Atropine and epinephrine have long been found useful in the allergies as have ephedrine and amphetamine.

Extracellular Fluid. Because the typical allergic wheal or edema is produced primarily by increased capillary permeability with the consequent formation of an area of extracellular fluid collection, drugs acting to decrease capillary permeability and to get rid of extracellular fluid have been found useful in the medical treatment of the allergies. Allergic edema has been shown by Rössle[86] to differ from cardiac edema only in its increased content of serum proteins. Schemm[93] found that extracellular edema in cardiac failure could be relieved by the elimination of sodium. Therefore, a low salt diet and diuretics have been found useful in treatment of the allergies both by Stoesser and Cook[99,100] and Kern.[54]

The Effect of Relative Acidity. Kuntz[58] stated that changes in the autonomic functional balance associated with changes in the acid-base balance have been amply demonstrated. Consequently restoration of the autonomic balance by appropriate therapeutic measures designed to restore the acid-base balance should not be regarded as beyond the range of possibility in allergic disease.

Alden,[3] Beckman,[11] Roberts[85] and others have reported success in the treatment of allergic disease by the use of acidifying agents to reduce the potential alkalosis.

Selye[95] also advocated the use of acidifying salts in disorders apparently provoked by an excess of the "salt active corticoids" as opposed to the "sugar active corticoids" such as the compound E (cortisone) of Kendall. In Ménière's disease ammonium chloride in enteric-coated capsules containing 0.5 gm., 4 to 6 capsules being given during meals three times a day, appears to be useful when combined with vasodilator therapy.

Ascorbic Acid. It is suggested that in some individuals allergic reactions are produced by a relative depletion of the adrenal cortical hormones. That this may be owing to the lack of ascorbic acid, the precursor of corticoid hormone, is indicated by the fact that ascorbic acid has been reported to be effective in relieving the symptoms of allergy in some individuals. Ascorbic acid also has a direct effect on capillary permeability, its lack being associated with increased permeability and fragility of the capillary wall.

Vitamin P. These substances have been found necessary to complement the local action of ascorbic acid. The

absence of both ascorbic acid and vitamin P appears necessary to produce the symptoms of scurvy. Beiler and Martin[12] found that in the presence of ascorbic acid, compounds having a vitamin P activity manifest a well-marked inhibitory action on hyaluronidase. These authors were unable to demonstrate whether this action was a direct inhibition of hyaluronidase by ascorbic acid or was due to a potentiation of vitamin P by this substance.

I have found the combination of ascorbic acid and rutin effective in certain manifestations of allergy, especially as a maintenance therapy to be taken after the acute symptoms of the disorder have been relieved by other medication. In my hands 250 mg. of both rutin and ascorbic acid taken three times a day tends to prevent the return of allergic symptoms. Saylor[92] recently has reported the effective treatment of allergic vasomotor rhinitis with hesperidin chalcone sodium.

Vitamin Therapy. Selfridge[94] has emphasized particularly the effect of the vitamins of the vitamin B complex on lipoid metabolism and vascular function. I have given vitamin therapy a trial but I have rarely found evidence of a marked change in a patient's symptoms or signs following vitamin therapy.

Fatty Acids. Hansen[43] has shown that lack of unsaturated fatty acids in the diet may lead to allergic conditions. Hansen found that certain infants with eczema were materially benefited when fats such as lard, corn oil and raw linseed oil were added to the diet.

I have observed marked relief to symptoms of vasomotor coryza, in children who refused to eat the fat of the meats served, by the feeding of the fats recommended by Hansen.

Specific Allergic Therapy. A carefully taken history will often reveal more clinically useful information in regard to foods, contacts and the like than skin tests. In pollenosis and inhalant allergies in general, however, useful clinical information may often be gained by skin testing. When clinically significant positive results to skin tests are obtained, attempts at hyposensitization are frequently beneficial. I have found, however, that supplementary medical treatment will often hasten and increase the symptomatic improvement even of patients who appear to be receiving clinical benefit from so-called specific therapy. The detail of successful specific management of the allergies forms a literature in itself and will not be considered here. Possibly the best outlines of treatment from the immunologic viewpoint for conditions in the ear, nose and throat are those furnished by Ashley,[5] Hansel,[40,42] Black[15] and Rawlins.[80]

SUMMARY

A theory of allergy based on the autonomic vascular reactions is felt to furnish a better working hypothesis from the standpoint of clinical diagnosis and treatment than one based on the antigen-antibody concept. At present the diagnosis of allergy is made clinically and positive reactions to skin tests merely give confirmatory evidence of allergy. In many such individuals positive skin reactions cannot be obtained. In the entire group, however, it is possible to obtain confirmatory evidence of allergy by studying the capillary bed by biomicroscopy. Treatment by specific methods leaves much to be desired. Supplementary treatment by nonspecific methods will often produce a favorable clinical result unobtainable by specific methods alone. There is evidence that the typical histologic picture of allergy may occur without the intervention of an antigen-antibody mechanism. A concept of allergy as localized or focal autonomic dysfunction is far

more consistent with clinical practice than the antigen-antibody concept; it opens up new avenues of therapeutic approach to the allergies and offers greater hope of symptomatic improvement to a patient who has one of the allergies.

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DISCUSSION

REA ASHLEY, M.D., San Francisco, Calif.: Dr. Williams' excellent paper reviews and analyzes much of the important allergic literature which has accumulated over the past fifty years. Upon the basis of his analysis and upon his personal experience he presents some of the newer concepts of allergy.

I am in entire accord with these newer ideas and believe, as he does, that all allergic reactions cannot be explained according to the antigen-antibody theory of immunity. In fact, I believe it most unfortunate that the terms "antigen," "antibody" and "immunity" should ever have been applied to allergy, since it has never been shown that allergy and infection bear any relationship whatsoever. The reactions of the body to allergic stimuli are entirely different from those produced by infection. Infections are the *cause* of cell injury. Allergic reactions are the *result* of cell injury.

Today, instead of considering allergy an immunologic phenomenon caused only by contact with poisonous proteins, it is believed that the allergic response is simply the increasing of a normal physiologic process—namely, relaxation and dilatation of the small blood vessels—and that the so-called "immunologic" mechanism is only one of the mediators of allergy. Direct trauma and so-called psychogenic stimuli can also initiate allergic attacks.

While Dr. Williams and I are in agreement as to the broad concepts of allergy, I should like briefly to emphasize some of the points he has made and to present certain other phases of the subject from a slightly different viewpoint.

The normal basic physiologic mechanism of the body is the constant state of contraction and dilatation of the small blood vessels with its attendant effect on the blood vessel walls and the shift of fluid content from one area to another. Through this mechanism the cells receive nourishment and dispose of waste products.

The small vessels are regulated in their state of dilatation and constriction by three general sets of forces: (1) nervous impulses, (2) chemical stimuli (the usual products of cellular life, the products of glandular secretions and other environmental factors) and (3) physical agents—heat, cold, light and mechanical irritations. We do not know whether all of these forces act through the same agency or independently, but we do know that the small vessel tone is maintained in a delicate balance by these forces and that the most minute changes in any one of them will affect this balance.

In an allergic reaction the normal body sympathetic-parasympathetic balance is thrown out of balance, with the *parasympathetics* gaining dominance over the *sympathetics*. Extreme dilatation of the capillaries results, causing capillary walls to become overpermeable and thus allowing excessive amounts of serum and electrolytes to escape into the surrounding tissue, causing edema. *Edema is the primary cause of symptoms in all allergic reactions,* and the character of the symptoms is determined according to the organ or tissue in which the swelling takes place.

It is my belief that allergic manifestations depend entirely upon disruption or imbalance of the autonomic nervous system caused by a lowered autonomic threshold of tolerance to alarm stimuli to which the autonomic system is constantly being subjected. These stimuli may arise from within the individual or from his environment.

The tendency to imbalance or the lowering of the autonomic threshold of tolerance may be influenced by heredity or may be acquired. Frequently both factors operate.

The distinction between allergic persons and those who are not allergic can be explained by the fact that no two individuals are identical in any respect and that the same variation which exists between individuals is also present in the individual cell's *reactivity* to stimuli and to *injury*.

In those individuals in whom cellular activity is great, hereditary allergy is apt to appear, while in those in whom it is of lowest degree, symptoms of allergy may never occur. Between these two extremes of varying degrees of cellular activity there exist all degrees of allergic symptom probability.

The threshold of autonomic tolerance based on cellular activity is individual and is the primary factor which determines who shall develop allergic symptoms.

Regardless of what the initiating factor or factors may be—house dust, chicken feathers, shell fish, insect bites, sunlight or emotional stress—the stimuli travel over the same pathways and as an end result produce the same type of allergic reaction.

In general three main types of allergic stimuli are recognized.

First, those proteins, protein-like and protein-attached substances which come in direct contact with the shock organs. In this group are included the inhalants, the ingestants, the injectants, the contactants, the infectants, and the infestants. This is the largest group and the allergen-reagen reaction is present. Hyposensitization is frequently possible in this group.

Second, the emotional stress group, which includes fear, anger and anxiety. It is thought emotional stress initiates allergic manifestations by direct action on the sympathetic-parasympathetic balance, upon the thyroid-pituitary-adrenal glands, and through direct action on the vasomotor center in the brain stem. The allergen-reagen phenomenon is not present and hyposensitization is not possible.

Third, physical agents such as heat, cold, sunlight and mechanical irritations which act directly on the small vessels and indirectly through the same pathways as the emotional group. The allergen-reagen phenomenon is not present and hyposensitization is not usually possible.

Any two or all three of these groups may, and often do, coexist and all must be taken into consideration when outlining treatment.

In our experience, medical treatment through the use of drugs, vitamins, hormones and other medical therapeutic agents is helpful especially in controlling acute allergic symptoms, but to date none of these therapeutic measures have been shown to alter permanently the autonomic threshold of tolerance, i.e., to cause the sympathetic-parasympathetic balance to become more stable. Hyposensitization alone does this, and while it is not always successful, it is nevertheless the most effective treatment which we have today for a large proportion of allergic reactions.

I wish to compliment Dr. Williams on the excellence and the timeliness of his most interesting and thought-provoking paper. As a basis for further investigative work, it is a real contribution and warrants careful reading and study.

JOHN M. SHELDON, M.D., Ann Arbor, Mich.: First I should like to congratulate Dr. Williams upon his most excellent presentation. I do not believe that we can take exception to his splendid concept, since we all appreciate the inadequacy of applying the antigen-antibody theory in the treatment of all allergic patients. Perhaps in part such failure may be due to the result of our lack of sufficiently good tools or technics for demonstration of antibodies. On the other hand, I am inclined to agree with Dr. Williams that it is more likely that there are other mechanics for the explanation of symptoms in certain allergic people.

The experiments of Selye and a number of other workers studying the pituitary-adrenal physiology may throw some light on the mechanisms of reaction on all states of hypersensitiveness. Fundamentally parenteral administration of adrenocorticotropic hormone or certain adrenal steroids will effectively control the symptoms of any allergic patient regardless of type and including the types that we have just heard described. The mechanism of such symptom control may possibly be a blocking effect at the tissue cell level.

Possibly this may be related to enzyme activity or to some other chemical which prevents the chain of events normally produced by a number of stimuli in allergic persons. You are all familiar with the fact that a multiplicity of stimuli may affect at different points this adrenal-pituitary axis. Perhaps stimuli arising in the higher motor centers through the hypothalamus may stimulate the pituitary gland, which in turn secrets adrenocorticotropic hormone, which in turn has an effect on the adrenal steroids. The opposite effect may occur, inducing symptoms as well as initiating symptoms.

On the other hand, certain stimuli may initiate the secretion of adrenalin, which in turn stimulates the pituitary, and the circle continues. The opposite effect may also occur.

Further study along this line may further clarify the concepts presented today. At least such mechanisms explain why psychic trauma, physical agents, antigens and a multiplicity of stimuli will initiate allergic symptoms, and conversely can explain relief of symptoms from a multiplicity of approachs such as we have heard today. Unfortunately, administration of substitutes such as adrenocorticotropic hormone or adrenal steroids is not the answer to clinical management and, therefore, it appears to me that Dr. Williams' concept is of great clinical value.

I am glad that Dr. Williams has emphasized that there are those individuals who have allergy related to antigen-antibody mechanism. As we emphasized a year ago at this meeting, it is imperative to demonstrate the antigen in this group of people if we are to obtain benefit by treatment with an antigen. In those patients in whom allergen cannot be demonstrated, treatment along epidemiologic lines will be of no avail.

I believe that Dr. Williams has given us a greatly improved outline for the care of allergic people, and I have greatly appreciated the opportunity of reading and listening to his paper.

DR. WILLIAMS: I am afraid I must apologize to Dr. Ashley for a certain lack of clarity in my presentation. The principal thing which I tried to suggest was that the body uses the same stereotyped resistance mechanism in resistance to stress of all types, which consists fundamentally of arteriolar constriction with a secondary dilatation of the capillaries and venules. Even antigen-antibody reaction appears to be secondary to this fundamental reaction. I am sorry that Dr. Ashley did not note that this was my fundamental concept.

I want to thank Dr. Sheldon for his very kind treatment of this paper, and I hope that some of you in reading it will get some benefit as far as treatment of your clinical patients is concerned, because even if an antigen-antibody reaction is present, this should not prevent physiologic therapy. I think there is very little more to say.

DIFFUSE EXTERNAL OTITIS: ITS PATHOLOGY AND TREATMENT

BEN H. SENTURIA, M.D.
ST. LOUIS, MO.

DIFFUSE external otitis, which masquerades under many other names, has been selected from the conglomeration of diseases of the external ear for discussion because it has been one of the least understood symptom complexes of the ear canal. Due to its uncertain pathogenesis, treatment has been diverse, unreliable and often ineffective.

In order to understand this disease entity, let us review this problem as presented in both textbooks and literature. Bezold and Siebenmann[4] wrote that chemical and mechanical influences must be named among the causes and emphasized the importance of the entrance of water into the canal. They added that "the real causa efficiens in all different forms of this disease are microorganisms, especially saprophytes." Politzer[27] stated that "the cast off epidermic scales must be microscopically examined for micrococci or for the aspergillus fungus." Kerrison[18] and Morrison[23] described only cases secondary to trauma and purulent drainage from the middle ear. W. Gill in "Diseases of the Nose, Throat and Ear" by Jackson and Jackson[17] stated that the most frequent causative organisms are fungi, but noted that "a resistant form of infection is said to be produced by the Pseudomonas pyocyaneus." Brown in Fowler's "Medicine of the Ear"[12] stated that the invading organism is usually a Streptococcus or may be the Pseudomonas, Plaut-Vincent or diphtheria organisms. Lederer[20] wrote of a low virulence Staphylococcus pyogenes which infects the hair follicles and sebaceous glands. Boies[7] spoke of a diffuse inflammatory process in which the causative organism is usually a Streptococcus or a Pseudomonas aeruginosa.

A brief review of the literature reveals that W. D. Gill,[14] Whalen,[42] Dart,[10] Trexler,[41] and McBurney and Searcy[21] emphasized the importance of fungi in the ear canal. Greaves,[16] Syverton,[40] Quayle,[29] Bettington,[3] Senturia,[30] Conley,[8] and E. K. Gill[13] emphasized the high incidence of gram-negative bacilli in cultures obtained from the external ear. Swimming and bathing have been described as contributory causes of this disorder,[1,8,19] while many authors[9,22] point out that a high percentage of sufferers are nonswimmers. Morley,[22] Palmer,[25] Daggett,[9] and Senturia[30] made reference to hot weather as a possible contributing factor.

Some attention has been directed to the character of the secretion. W. Gill[15] and Conley,[8] among others, noted the absence of cerumen. Daggett[9] was impressed with the "desquamated debris" which he described as consisting of epithelial scales, pus cells and organisms. Senturia, Matthews and Adler[35] noted the myriads of epithelial cells and bacteria and the remarkable absence of neutrophils in this form of external otitis.

One comes away from such a review in much confusion and with many un-

From the department of otolaryngology, Washington University Medical School, St. Louis, Mo.

This research was aided by grants from the Research Study Club of Los Angeles, Calif., and the St. Louis Otological Foundation of St. Louis, Mo.

Presented at the Fifty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 8-13, 1950, Chicago, Ill.

answered questions. What is the pathogenesis of diffuse external otitis? What is the role of fungi? Why are gram-negative bacilli found in such a high percentage of cases? Why do smears of the secretion reveal no neutrophils?

In order to avoid any uncertainty as to the disease entity which is being discussed, let us describe the clinical picture which has been designated as diffuse external otitis. This is a specific symptom complex occurring mainly during hot, humid weather. It may occur as a mild, moderate or severe disease and may be acute or chronic according to the symptoms and findings.

The mild acute case has only slight pain on mastication or on manipulation of the auricle. A thin watery secretion may be found in the lumen. The skin of the ear canal reveals some edema, slight redness, and a coating of odorless, adherent secretion or exfoliated debris. Some loss of luster of the drum is seen.

In the more severe cases patients complain of intense throbbing pain on mastication and on manipulation of the external ear. Although the auricle appears uninvolved, there is marked periauricular edema and partial or complete obliteration of the canal lumen by the edematous walls. Gray or green serous or "seropurulent" secretions and sheets of exfoliated debris are seen in the remaining lumen. The skin of the canal is thickened, purplish red in color, and may have a papular or "gooseflesh" appearance, particularly on the superior and inferior walls. There is, characteristically, a smooth, convex sagging of the superior canal wall extending to the tympanic membrane. Under otoscopic magnification, discrete, raised, milky white papules and flat grayish vesicles are seen with intervening zones of erythema. Because of the obliterated lumen the tympanic membrane cannot be well visualized.

The chronic case reveals a variable thickening of the skin of the auricle and ear canal and a consequent reduction of the lumen of the entire canal. Dry, adherent, exfoliated debris often lines the canal, or there may be gray-brown or greenish secretion, with a fetid odor, coating the skin and filling the tympanic recess. Papules and vesicles may or may not be seen. The drum is lusterless, thickened and shows loss of some detail.

Cultures of the ear canals almost invariably show an overgrowth of gram-negative bacilli, while occasional fungi are seen. A stained smear reveals myriads of bacilli and epithelial cells.

Now that the entity with which we are dealing has been described, let us briefly designate its place in relation to the many other forms of disease involving the external auditory canal. If we divide the cases on an etiologic basis, we may classify them as follows:[34]

Diseases of the External Ear

I. Malfunction of skin glands (etiology unknown)
   a. Diffuse external otitis
   b. Seborrheic dermatitis
II. Infections
   a. Bacterial
   b. Fungus
III. Neurogenic
IV. Allergic
V. Primary irritants
VI. Senile changes
VII. Endocrine dyscrasias
VIII. Vitamin dyscrasias
IX. Miscellaneous

In order to understand the pathogenesis of diffuse external otitis we must first examine the skin of the normal external auditory canal (fig. 1).[33]

In the normal skin there is a constant exfoliation of the upper layers of the stratum corneum. The sebaceous and apocrine sweat glands discharge their secretions over the surface of the skin to form a thin, fatty, protective coating

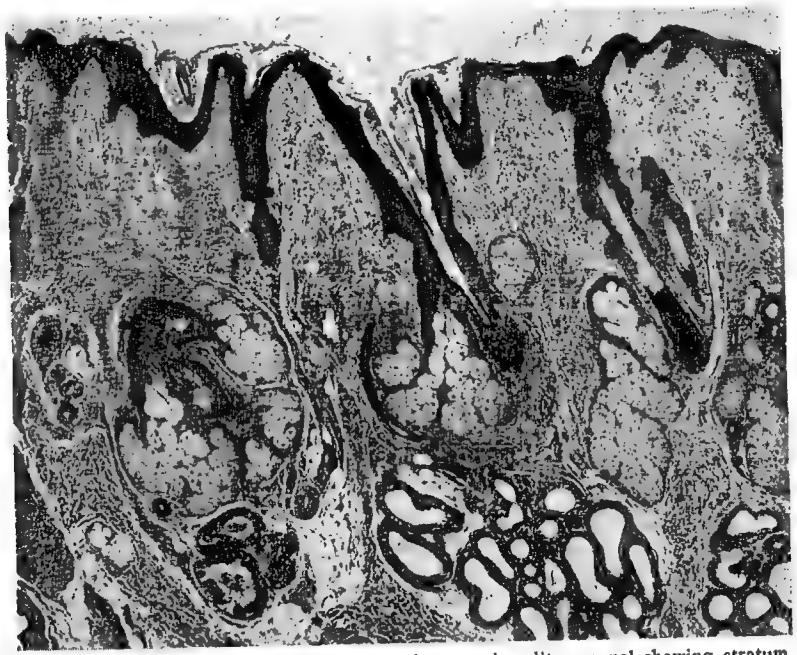

FIG. 1—Photomicrograph of skin of normal external auditory canal showing stratum corneum, sebaceous glands, hair follicles and apocrine glands.

with a slightly acid pH.[5,11] These elements, which in the ear canal combine to form the cerumen, have been shown to have a bacteriostatic and fungistatic action in other parts of the body.[2,6,26] A culture of these secretions coating the normal skin of the ear canal shows some gram-positive bacteria, rarely a fungus.[32] A smear reveals occasional epithelial cells and bacteria.[35]

If, now, the normal skin is traumatized or irritated by the prolonged application of macerating or noxious substances, there is produced an increased hyperkeratosis and plugging of the pilosebaceous follicles and ducts of the sweat glands.[24,28,39] These pathologic changes have been produced in experimental work on prickly heat by the prolonged application to the skin of water, adhesive tape,[36,37,38] chemicals,[24,37] etc. It is seen in biopsy specimens of the skin obtained from the ear canal in cases of chronic otorrhea.[33]

It is conceivable that as a result of prolonged exposure to intense heat and humidity and poor evaporation within the ear canal, the skin of the ear canal is bathed in apocrine sweat which is relatively low in lipoids. The unprotected upper layers of the stratum corneum imbibe the water, become swollen and macerated, and proper cornification does not occur.[28,39] As a consequence, marked hyperkeratosis develops (fig. 2).

If, at this time, the patient avoids further exposure to high temperatures and humidity, the hyperkeratosis, as demonstrated in other skin areas,[38] will disappear in approximately two to three weeks as a result of normal desquamation.

On the other hand, if before this hyperkeratosis subsides there is another prolonged exposure to high temperatures and humidity or the patient traumatizes the skin surface, the following events may occur:

A. As a result of the obstruction of the ducts of the sweat glands and pilo-

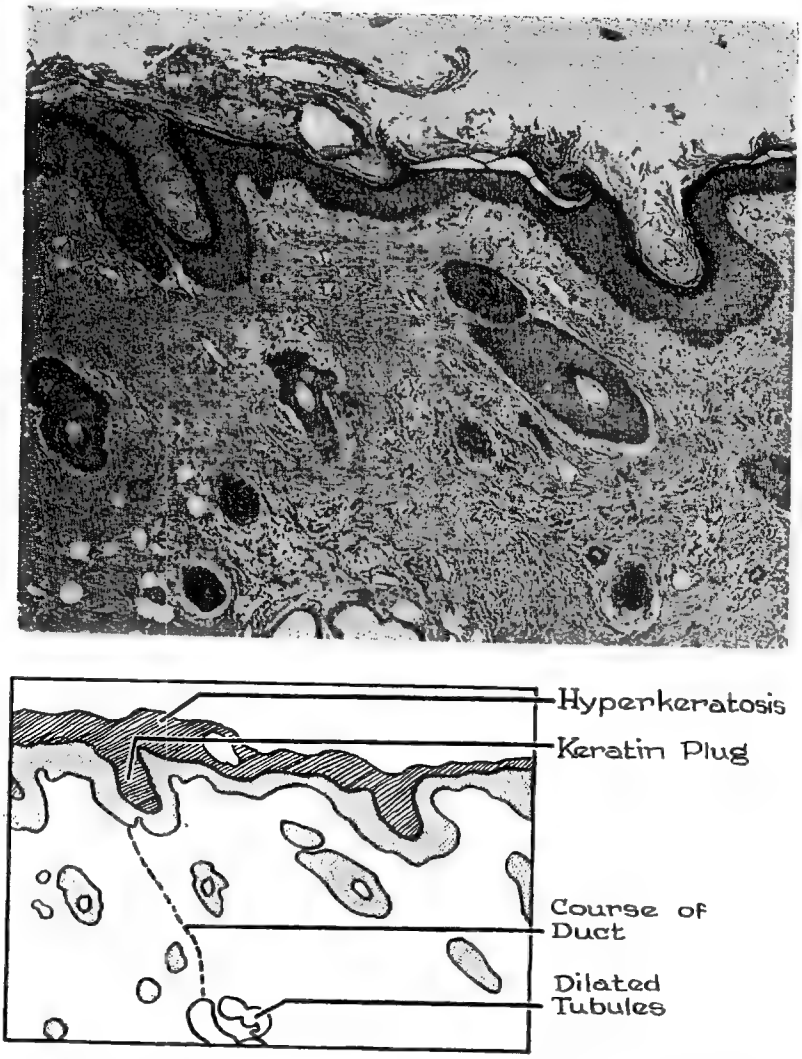

FIG. 2—Photomicrograph of skin of external auditory canal showing hyperkeratosis and plugging.

sebaceous follicles, the antibacterial and the antifungus elements are not secreted onto the skin surface of the canal and therefore bacterial or mycotic growth, or both, may occur. This growth would be determined by the transient flora present in the environment or introduced by the patient. In tropical areas there would be a relatively high incidence of fungi; in temperate zones fewer fungi would occur. In all parts of the world the widely disseminated gram-negative bacilli would be cultured.

B. There may be thickening of the prickle cell layer (acanthosis), retention of nuclei in the stratum corneum (parakeratosis), intracellular and intercellular edema. A dense cellular infiltrate may occur in the subepidermal areas and occasionally around the tubules of the sweat glands. Marked dilatation of lymphatics and blood vessels,

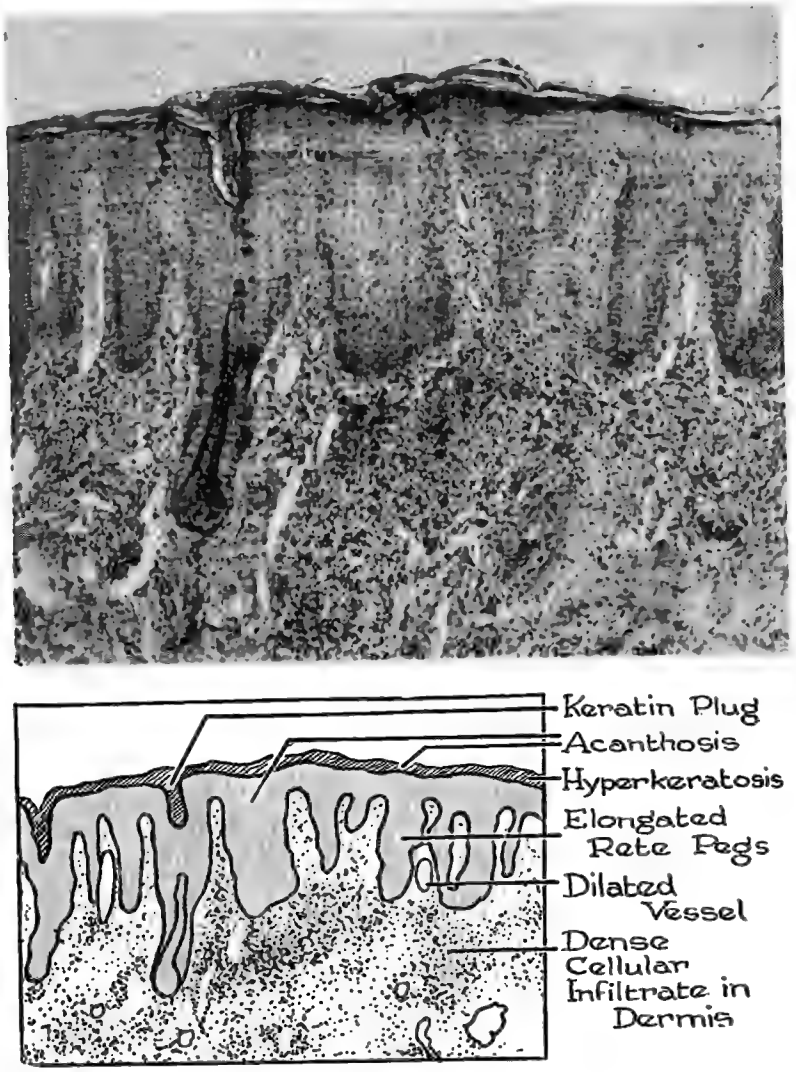

FIG. 3—Photomicrograph of skin of external auditory canal showing hyperkeratosis, acanthosis, lengthening of rete pegs, dilatation of blood vessels and lymphatics, and dense cellular infiltrate in dermis.

and collection of fluid in the interstitial spaces of the dermis may be observed (fig. 3).

C. There may be dilatation of the apocrine sweat ducts as a result of plugging of the ducts and possibly a diffusion of the retained sweat into the epidermis (fig. 4).[24]

D. There may also occur changes in the tubules such as dilatation, and vacuolization of the epithelial lining cells (fig. 5).

E. Finally, as a result of the inflammatory process plus the failure to eliminate the obstruction of the ducts there may occur a disorganization of the epithelium of the tubules (fig. 6) and a destruction of many of the sweat glands (fig. 7).

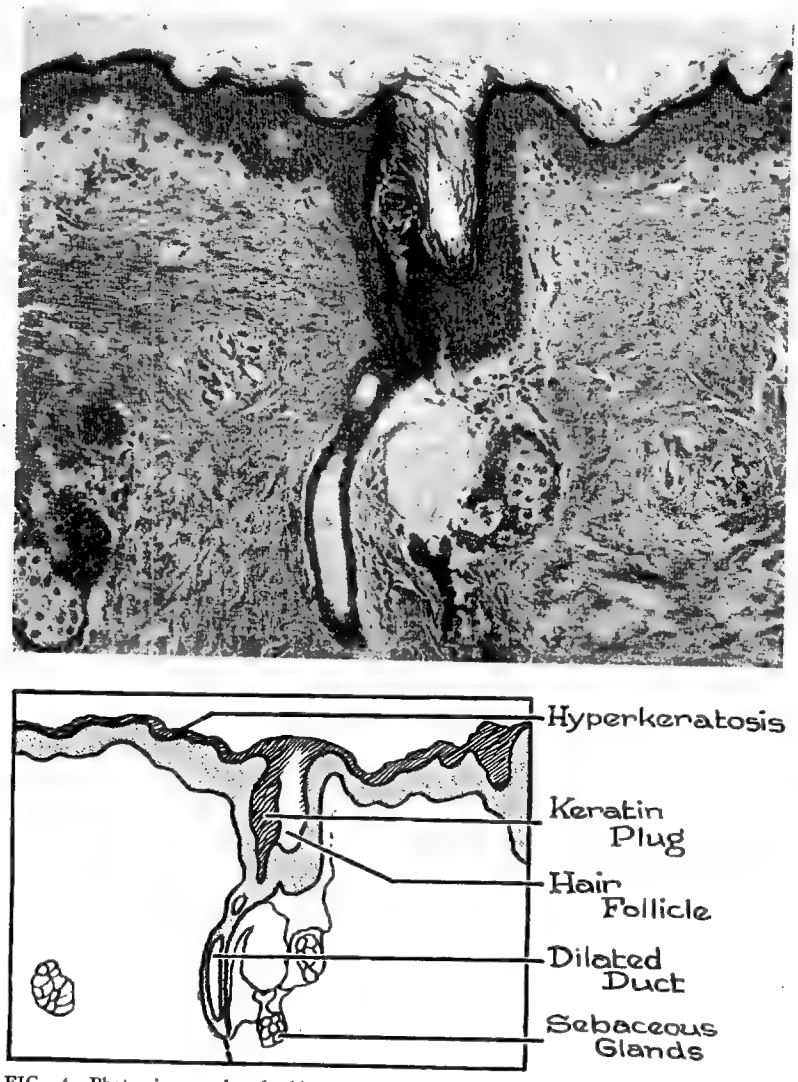

FIG. 4—Photomicrograph of skin of external auditory canal showing obstructing keratin plug and dilated duct.

To recapitulate, although all the pathologic evidence has not yet been obtained, it is my belief that the preceding findings justify the following concept of the pathogenesis of diffuse external otitis. As a result of excess sweating during hot, humid weather there is produced in certain susceptible individuals a marked hyperkeratosis of the skin of the ear canal with plugging of the ducts of the sweat and sebaceous glands. If recovery is not allowed to occur before another period of exposure to a hot, humid environment, these glands are unable to pour their secretions onto the skin. As a consequence of the skin changes produced, a surface infection may readily develop. If the plugging remains and sweat gland activity persists, there occur inflammatory and mechanical changes within the epidermis and dermis.

Thus diffuse external otitis is not caused by swimming or bathing, although these may be factors in producing the increased hyperkeratosis and plugging of the glands which initiate the pathologic cycle of events. It is not caused by fungi or bacteria but rather these are secondary invaders resulting from the loss of skin resistance. Neutrophils are not found on smears of the flow of secretions from the apocrine sweat glands and sebaceous glands

Treatment, then, should be soothing and palliative during the acute stage while active, and curative in the subsiding stage. Thus therapy might be directed as follows:

A. Palliative

1. Remove the patient from the hot, humid environment. This will reduce the activity of the apocrine sweat glands and thereby alleviate much of the discomfort.
2. Irrigate the ear canal with hypertonic saline[31] in order to obtain drainage and to clean away the keratin plugs, inspissated wax and debris which have collected in the ear canal and tympanic sulcus.
3. Apply into the lumen of the canal a mild, nonirritating antiseptic in a fatty vehicle (e.g., 5 to 10 per cent boric acid in anhydrous lan-

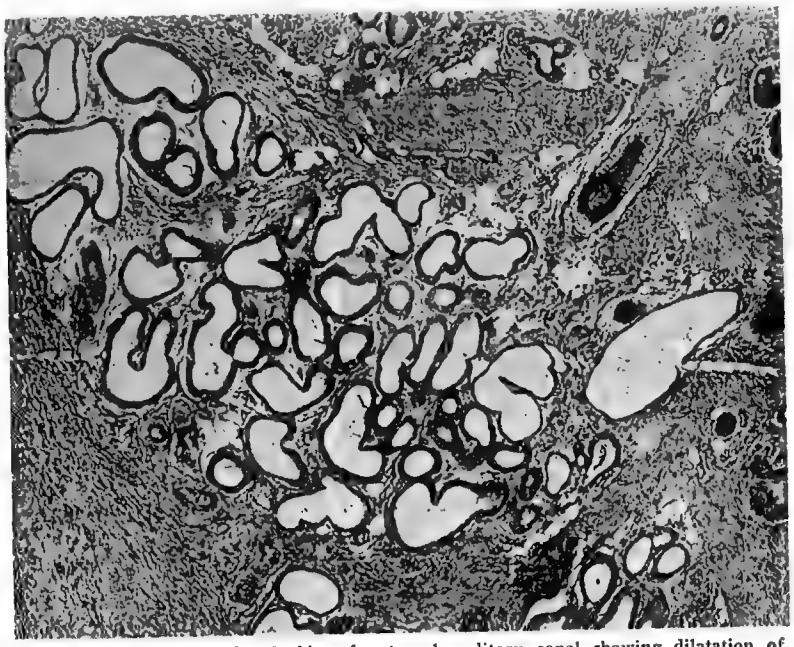

FIG. 5—Photomicrograph of skin of external auditory canal showing dilatation of tubules and vacuolization of lining cells.

secretion since we are not dealing primarily with a pyogenic infection breaking through the skin barrier except in the complicated cases.

If we accept this concept of the pathogenesis of diffuse external otitis, therapy, in order to be successful, must accomplish three things:

1. Counteract the secondary infection
2. Restore the depleted lipoid substances of the skin
3. Modify the stratum corneum which is acting to obstruct the normal

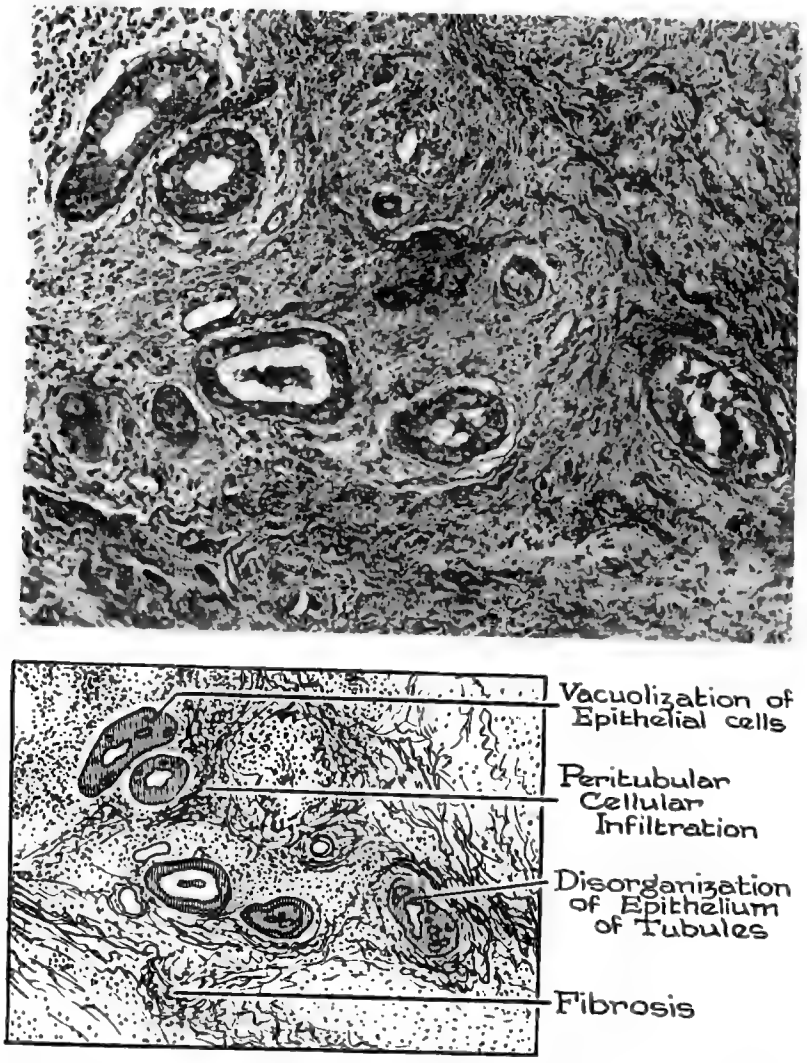

FIG. 6—Photomicrograph of skin of external auditory canal showing disorganization of the epithelium of the sweat gland tubules.

olin) and allow to remain in place for 12 hours. This will restore the depleted lipoids and will also tend to push the pH towards the acid side, thus aiding in the inhibition of bacterial growth.

4. Prescribe antibiotics where specifically indicated.
5. Give vitamin A to assist in the restoration of normal keratinization.

B. Curative

As soon as the severe acute stage has subsided, active attempts should be directed towards eradicating residual infection and restoring the normal stratum corneum. This may be accomplished with the aid of the following medicaments according to the severity of the residual inflammatory reaction and the sensitivity of the skin.

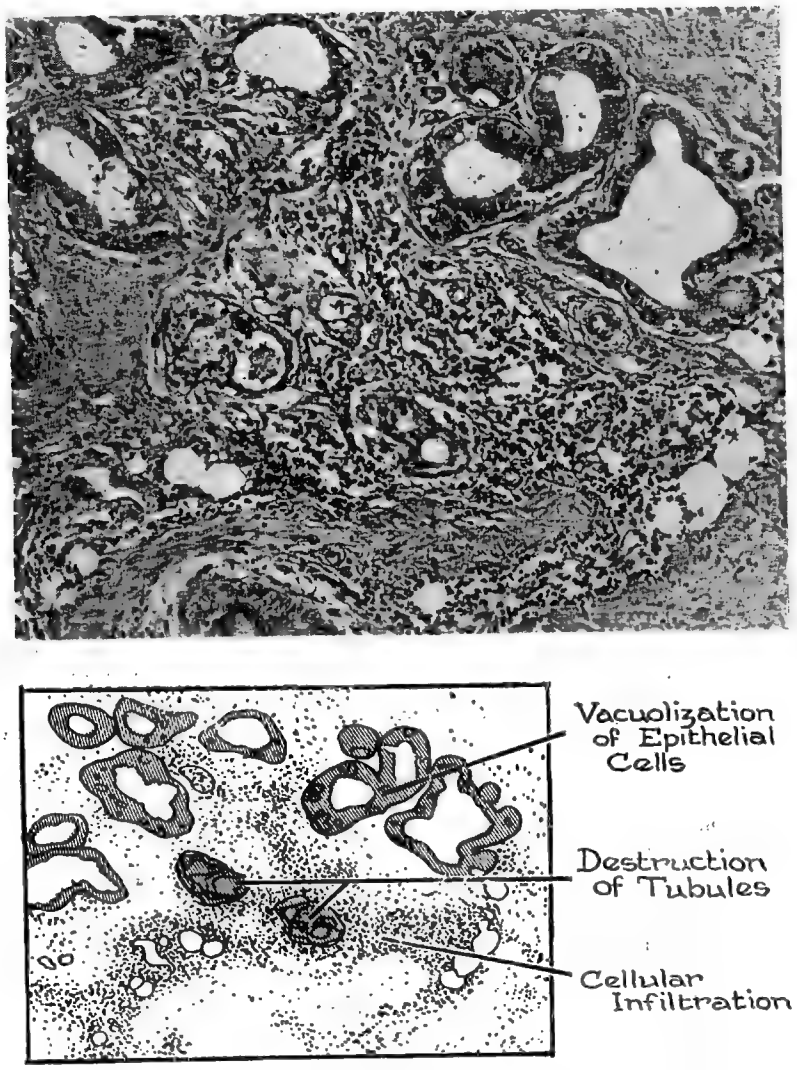

FIG. 7—Photomicrograph of skin of external auditory canal showing destruction of sweat gland tubules and peritubular cellular infiltration.

1. Burow's solution, 0.5 per cent, or similar stable proprietaries
2. Metacresylacetate in olive oil (one-half strength)
3. $AgNO_3$, 5 to 10 per cent, followed by 5 to 10 per cent boric acid in anhydrous lanolin
4. Streptomycin (5mg/gm) in a carbowax vehicle[32]
5. Vitamin A — 10,000 to 20,000 units per day

Astringents should be continued for 24 to 72 hours if the agent is tolerated. The natural healing processes of the skin should then be allowed to take over. Further therapy should consist of adequate cleansing of the skin surface with hypertonic saline and the application of soothing antiseptic ointments, keratolytics, fungicides or antibiotics as indicated by individual requirements.

I wish to express appreciation to Dr. Zola Cooper for advice and assistance in the pathologic studies, to Mr. Vernon Fischer and Mr. Wallace Johnson for technical assistance, to Mr. K. Cramer Lewis for photomicrographs, and to Mr. F. Kelly for diagrams.

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DISCUSSION

FRANCIS E. SENEAR, M.D., Chicago, Ill. (by invitation): This has been a very interesting paper to me because this problem comes to us very frequently. The etiology of otitis externa has, of course, been the source of controversy for many years. It has been our impression that until the past few years the majority of the otologists have emphasized chiefly the possibility of mycotic infection as the most common cause, since patients whom we have seen after they have previously consulted otolaryngologists have usually stated that they have been told their trouble was due to fungus infection, usually without any microscopic examination having been made.

Dermatologists have in the main been unwilling to accept this concept due to the fact that the fungi demonstrable in the ear were usually of nonpathogenic varieties and rarely of types giving rise to the usual dermatologic disorders of mycotic origin. We, on the other hand, have been accustomed, I think, to incriminate seborrheic dermatitis and streptococcic infections as being responsible for most of these cases, such diagnosis usually being made, likewise, on purely clinical grounds. In recent years, however, the literature of your specialty has minimized the importance of fungus infection, relegating it to an infrequent role and perhaps then considering it as only a secondary factor. Probably most often it is regarded as being only a saprophyte. Consequently a number of studies have pointed out that a variety of bacteria are of etiologic importance. These opinions with regard to the organisms responsible for diffuse otitis externa have not, however, given us any idea as to the basic pathogenesis of the process.

A month or two ago, in looking through my files on eczema, I ran across an abstract I had made of an article by Dr. Senturia published in 1946, in which he discussed etiology from the fungus and bacterial standpoints, with nothing at all pertaining to what he has given us today. He is to be congratulated on having introduced a new idea in the study of this condition.

We in dermatology during the second world war became aware of the importance of hyperkeratosis as it developed at the follicular orifices in the tropics during periods of excessive sweating, leading to suppression of perspiration through retention rather than the failure of the glands to produce sweat, namely, sweat retention anhidrosis. Likewise in recent years we have recognized that in acne vulgaris, the disturbance in keratinization resulting from the disturbance of the pilosebaceous apparatus in adolescence is of primary importance in preparing the way for pathogenic organisms to produce the pustular lesions of disease.

Dr. Senturia has presented a careful, well documented study of this problem, and while, as he states, all the pathologic evidence has not yet been obtained, his application of this principle that hyperkeratinization furnishes a soil of lowered resistance upon which organisms may flourish and produce the pathologic changes, opens a new avenue. I know that he is continuing his studies in this field, and I am sure that his work will be received grate-

fully by those in dermatology. The illustrations which he showed you on the screen today and which I had the privilege of examining in the photomicrographs lend excellent support to his thesis that we are going to have a very good basis upon which to proceed in the future.

I have enjoyed this paper very much and want to thank Dr. Senturia.

W. D. GILL, M.D., San Antonio, Texas: I am very much impressed with this paper. I think it is one of the best presentations on the subject that I have ever heard. I think also that I am safe in saying that this is the first time in the history of otomycosis or diffuse external otitis that we have seen any histopathologic sections demonstrated. That is a great step forward. There is a great deal to be learned from the study of the fixed tissue cell in this condition, and Dr. Senturia has pointed the way for us.

There has been a great tendency in the past few years to minimize the importance of fungi in external otitis. I am not quite ready to agree with that thesis—with that hypothesis. I believe there has been a marked decline in the number of fungus infections in the external auditory canal. Just why that has come about has not become immediately apparent, but we know that there has been in our section, at least, a very marked decline in the percentage of fungus infections in the ear, with a corresponding increase in the Actinomyces and Staphylococcus.

(Slide) My brother, Dr. King Gill, and I recently had occasion to study the bacteriologic flora in the ears of 168 patients with the following results: We isolated bacteria in 117; bacteria mixed with fungi in 36; bacteria and yeast in 12; and bacteria, yeast, and fungi in 3. To get the total number of ears in which fungi were present, it is necessary to add those last three figures together.

(Slide) In a further breakdown, we found a very high percentage of gram-negative bacilli —104 cases. Those are the Pseudomonas, and at the present time these cases are in the ascendency. Then we had 6 cases due to staphylococci. I think that the incidence of Staphylococcus is certainly high enough to make it a very important factor in the etiology of diffuse external otitis.

In cleaning the ear I have always just used a sterile swab rather than irrigation. Occasionally when an ear is irrigated there will be a marked flare-up with suppuration following, and where it has been necessary to irrigate an ear to remove plugs of various kinds of debris, we have immediately dried the canal and inserted a wick moistened with 1:100 neutral aqueous solution. That seems to prevent the onset of suppuration.

Now a word about metacresylacetate. The pseudomonas respond to drugs containing acetic acid or the acetate radical; therefore, Burow's solution is effective. Two per cent aqueous solution of acetic acid is also effective, as well as cider vinegar and metacresylacetate. Metacresylacetate is bactericidal. It is fungicidal. It is analgesic after the first few minutes. It is also anesthetic and possesses varying degrees of keratolytic power, so that the top layer of the epithelial cells is removed merely by inserting a wick moistened with metacresylacetate and allowing it to remain 24 hours. The patient is asked to return at the end of that period, the wick is removed, and another one is inserted after cleaning the ear.

The ear may be so acutely inflamed that it tolerates practically no manipulation on the patient's first trip to the office. When that takes place, it has been our practice gently to induce a very small wisp of cotton into the canal and to saturate that with metacresylacetate. All of the severe cases immediately receive roentgen therapy. They are referred to the radiologist immediately, and the amount of x-ray that is given is one-third of an erythema dose. If both ears are involved, the dosage is divided equally between the two ears. When a patient has come in to see you because he is in pain, he wants quick relief. The metacresylacetate wick plus roentgen therapy is about the quickest way to get it.

I would implore you to use some type of sedative in all these cases that are at all severe. In this respect I should like to say that I consider codeine about the worst drug that can be used for sedation. In combination with some other drug it works fine, but alone it is practically worthless. One gram of codeine combined with 5 grains of amidopyrine, or 3½ grains of antipyrine, or 5 to 10 grains of aspirin are all good combinations and they work. But just remember that the patient has come in to see you because he wants relief and if you don't give it to him, Dr. John Jones across the hall will. At least I have found it that way.

Cultures have been made in studying the effect of a lot of the present-day fungicides that are on the market. There is a plethora of fungicides that are advertised today for the treatment of so-called fungus ears. We have studied a group of some ten or twelve, with the result that we have found most of

them to be practically useless. Sulfamylon was very effective, however. Except for the burning which it causes, it works very nicely. Streptomycin, we hesitated to use, internally at least, on account of the possibility of complications in the hearing apparatus.

I have enjoyed this paper very much. This is a subject that has been very close to my heart for about twelve years. I think we have made great strides forward, and now we are glad to have some studies on the fixed tissue cells.

Personally, I should like to ask Dr. Senturia where he gets his specimens for biopsy. I cannot obtain specimens from my patients. They object. How does he persuade them?

DR. SENTURIA: I should like to thank the discussers.

To answer Dr. Gill's question about biopsies, we have been very fortunate in having the cooperation of physicians on various city hospital services. We have had very wonderful patients who have been willing to provide us with material for biopsies. We have utilized material from autopsies and from mastoidectomies. Dr. Theodore Walsh and his staff have provided us with tissue from mastoidectomies performed at McMillan Hospital, St. Louis, during the past several years.

I should like to say one word about the incidence of otomycosis. At Randolph Field, Texas, we found approximately 10 per cent of infected cases showed fungi in the ear canal on cultural study of cases of external otitis. Despite this low incidence we have continued to feel that otomycosis is an important problem. If one neglects this consideration of fungus infestation of the ear canal, these patients will not get well. I should like to emphasize our belief that gram-negative bacilli are not the cause of diffuse external otitis. In a previous paper we noted a 100 per cent incidence of gram-negative bacilli in severe, acute, diffuse external otitis, but we were not able to say that gram-negative bacilli or Pseudomonas organisms were the cause of diffuse external otitis. We feel that the findings in this report tend to corroborate that impression. The gram-negative bacilli occur as a secondary infection. We believe that fungi are implanted secondarily.

This does not mean that the secondary infection is a simple problem which one may neglect. One may have to use every therapeutic aid available, on occasion, to eliminate that superimposed infection. We heartily agree with the use of metacresylacetate for otomycosis. We found, in our in vitro experiments, that it was a potent agent for eradication of saprophytic fungi. I would hesitate, however, to use it in the acute inflammatory state. Thus, I would imagine that Dr. Senear would shudder at the thought of applying as strong an agent as full strength metacresylacetate to the acutely inflamed ear canal of a patient with severe, diffuse external otitis. On the other hand, I am fully aware that one must offer the patient relief, and that sometimes one must throw caution to the wind and utilize an agent which may be irritating and caustic if it is to provide relief from pain.

We have investigated some of the acetates, with the thought in mind that we desired an agent with an acid reaction and that these appear to be active against gram-negative bacilli. Acetic acid is well known as one of the best agents against the Pseudomonas organism, but we advise its use thoughtfully and carefully in conjunction with other therapeutic aids.

I should like to take just one more moment to tell you about the use of irrigations for cleansing the ear canal. We have made serial cytologic studies of the secretions of the ear canals and have noted an absence of neutrophils in uncomplicated acute, severe, diffuse external otitis. We have observed the sudden appearance of neutrophils following the energetic cleansing of debris from the ear canal. Active, thorough cleansing is desirable, but not if you are going to traumatize the epidermis and push the infection into the subepidermal areas. Therefore, it is urged that saline irrigations be used to cleanse the ear canal. Exacerbations occur if one irrigates with water or even with isotonic saline. To avoid flare-ups, a hypertonic saline solution is suggested. For the past three years we have used, routinely, a 3 per cent saline douche of the ear canal in those cases where there is sufficient lumen so that the water can be removed and the ear canal dried.

In Memoriam

Ephraim Kirkpatrick Findlay.....Chicago, Illinois
September 27, 1950

James Sonnett Greene.....New York, New York
September 17, 1950

Marcus Ravdin.....Evansville, Indiana
September 12, 1950

Burt R. Shurly.....Detroit, Michigan
October 20, 1950

Edward McColgan Walzl.....Baltimore, Maryland
August 10, 1950

TRANSACTIONS
American Academy of Ophthalmology and Otolaryngology
Official Publication of the American Academy of Ophthalmology and Otolaryngology
Published under the direction of the Editorial Board
WILLIAM L. BENEDICT, Rochester, Minnesota, *Editor*
W. HOWARD MORRISON, Omaha, Nebraska, *Associate Editor*
HENRY L. WILLIAMS, Rochester, Minnesota, *Associate Editor*
Printed by Douglas Printing Company, 109 North 18th Street, Omaha 2, Nebraska

All communications regarding the TRANSACTIONS should be addressed to William L. Benedict, M.D., Executive Secretary-Treasurer, the American Academy of Ophthalmology and Otolaryngology, 100 First Avenue Building, Rochester, Minnesota

COUNCIL MINUTES

FIFTY-FIFTH ANNUAL SESSION

October 7 and 11, 1950
Palmer House, Chicago

Present:

J. Mackenzie Brown, Derrick Vail, Edwin B. Dunphy, Kenneth L. Craft, James Mason Baird, Alan C. Woods, Carl H. McCaskey, Conrad Berens, William L. Benedict, Algernon B. Reese, James H. Maxwell, A. D. Ruedemann, Dean M. Lierle, Lawrence R. Boies, Erling W. Hansen, Thomas D. Allen, Fletcher D. Woodward, Archie D. McCannel, C. Stewart Nash.

The meeting was called to order at 9:30 a. m. by Dr. J. Mackenzie Brown, President.

I

The Executive Secretary, Dr. William L. Benedict, reported on the membership as follows:

1. *Fellowship as of October* 1, 1950:

Juniors	3,907
Seniors	150
Life	427
Honorary	26
	4,510

2. *Candidates*:

Complete	280
Incomplete	7
	287

3. *Elevations to Senior Fellowship on January* 1, 1951:

Beal, Homer A. ..... Kansas City, Mo.
Birsner, Louis ..... St. Louis, Mo.
Bonner, William F. ..... Childress, Texas
Brown, Albert L. ..... Cincinnati, Ohio
Chapman, S. Jefferson, Colorado Springs, Colo.
Cleff, Oscar ..... Chicago, Ill.
Daily, Louis ..... Houston, Texas
Daily, Ray K. ..... Houston, Texas
Fox, Noah ..... Chicago, Ill.
Gipner, John F. ..... Rochester, N. Y.
Goar, Everett L. ..... Houston, Texas
Gordon, Charles H. ..... Portland, Maine
Hamlin, Fred E. ..... Roanoke, Va.
Hands, Sidney G. ..... Davenport, Iowa
Hansel, French K. ..... St. Louis, Mo.
Hargitt, Charles A. ..... Brooklyn, N. Y.
Harrell, Voss ..... Reno, Nev.
Hartshorne, Isaac ..... New York, N. Y.
Hicks, Vonnie M. ..... Raleigh, N. C.
Jones, Edmund L. ..... Wheeling, W. Va.
Joy, Harold H. ..... Syracuse, N. Y.
Kettelcamp, Fred O., Colorado Springs, Colo.
Key, Samuel N. ..... Austin, Texas
McLaurin, John G. ..... Dallas, Texas
McMahon, Bernard J. ..... St. Louis, Mo.
Minsky, Henry ..... New York, N. Y.
Morrison, W. Wallace ..... New York, N. Y.
Myers, E. Lee ..... St. Louis, Mo.
Packard, Louis A. ..... Phoenix, Ariz.
Palmer, Arthur ..... New York, N. Y.
Pember, Aubrey H. ..... Janesville, Wis.
Pendexter, R. Stevens ..... Washington, D. C.
Reincke, George F. ..... New Ulm, Minn.
Seal, J. Coleman ..... New York, N. Y.

Some items are omitted from this report. Complete minutes are on file in the office of the Executive Secretary-Treasurer.

Seligstein, Milton B. .....Memphis, Tenn.
Sharp, Benjamin S. .....Providence, R. I.
Sheahan, William L. .....New Haven, Conn.
Stokes, William H. .....Lake City, Mich.
Wagener, Henry P. .....Rochester, Minn.
Wright, Walter W. .....Toronto, Ont., Can.
Young, Charles A. .....Roanoke, Va.

4. *Elevations to Life Fellowship on January 1, 1951:*

Alden, Arthur M. .....St. Louis, Mo.
Bahn, Charles A. .....New Orleans, La.
Brickley, Daniel W. .....Marion, Ohio
Brown, Mortimer G. .....Syracuse, N. Y.
Buvinger, Charles W. .....East Orange, N. J.
Cohen, Samuel .....Philadelphia, Pa.
Darmer, George A. .....Aurora, Ill.
Dintenfass, Henry .....Philadelphia, Pa.
Hughes, T. E. .....Richmond, Va.
Husik, David N. .....Philadelphia, Pa.
Jordan, George T. .....Vermillion, S. D.
King, Edward Daniel .....Hollywood, Calif.
Lewis, Fielding O. .....Media, Pa.
Reese, Warren S. .....Philadelphia, Pa.
Schlanser, Adam E. .....Washington, D. C.
Williams, Walton H. .....Middletown, Ohio
Zentmayer, William .....Philadelphia, Pa.

5. *Deaths Reported since Last Council Meeting:*

Arnold, Francis J. .....Burlington, Vt.
Berry, David Franklin .....Indianapolis, Ind.
Black, William Byron .....Kansas City, Mo.
Brandenburg, Nora B. .....Winnetka, Ill.
Briglia, Frank Joseph .....Philadelphia, Pa.
Burke, Thomas Aloysius .....Cleveland, Ohio
Butterfield, Elwyn Temple, Las Vegas, N. M.
Carter, Albert Arthur .....Boston, Mass.
Charles, Joseph W. .....St. Louis, Mo.
Donoher, William David .....Los Angeles, Calif.
Durr, Samuel Abraham .....San Diego, Calif.
Fowlkes, John Winston .....New York, N. Y.
Friedenwald, Harry .....Baltimore, Md.
Fuller, T. E. .....Texarkana, Texas
Gale, M. Jean .....Denver, Colo.
Gradle, Harry S. .....Sherman Oaks, Calif.
Hewitt, Wright Platt .....Cambridge, Mass.
Higgins, R. P. .....Cortland, N. Y.
Hill, Emerson Stanley .....Torrington, Conn.
Howard, Joseph William .....Kansas City, Mo.
Howard, William H. .....Oelwein, Iowa
Hunt, Westley Marshall .....New York, N. Y.
Kayser, Reuben .....Brooklyn, N. Y.
Kutscher, Charles F. .....Pittsburgh, Pa.
Leavy, Charles A. .....Clayton, Mo.
Lingeman, Edward L. .....Indianapolis, Ind.
Lore, John M. .....New York, N. Y.
Matthews, Justus .....Minneapolis, Minn.
McAuley, Albert G. .....Montreal, Que., Canada
McDannald, Clyde Elliott, New York, N. Y.
McDowell, Nathan D. .....Rochester, N. Y.
McLaughlin, Roy Carlyle, Los Angeles, Calif.
Mengel, Sterling F. .....Pottsville, Pa.
Nance, Willis O. .....Chicago, Ill.
Neff, Eugene E. .....Madison, Wis.
O'Connor, Thomas P. .....Chicago, Ill.
O'Hora, James T. .....Detroit, Mich.
Parsons, Joseph G. .....Crookston, Minn.
Randel, Harvey O. .....Oklahoma City, Okla.
Ratner, Simon Harry .....Miami Beach, Fla.
Repass, Robert Eldon .....Miami, Fla.
Rindlaub, John H. .....Fargo, N. D.
Ringle, Charles A. .....La Junta, Colo.
Rubendall, Clarence .....Omaha, Neb.
Ryan, Maxwell D. .....New York, N. Y.
Schoofs, Orlando P. .....Milwaukee, Wis.
Spengler, John Arthur .....Geneva, N. Y.
Stueber, F. G. .....Lima, Ohio
Van Poole, Gideon M. .....Honolulu, Hawaii
Walker, Orville J. .....Youngstown, Ohio
Wells, David W. .....Newton, Mass.
Williams, Horace J. .....Philadelphia, Pa.
Woodry, Norman Lee .....Detroit, Mich.

6. *Resignations Received:*

Constans, George M. .....Bismarck, N. D.
Crowe, Samuel J. .....Baltimore, Md.
Finney, Nancy E. .....Cincinnati, Ohio
Gubner, Julius .....Brooklyn, N. Y.
Nebinger, Reid .....St. Petersburg, Fla.
Neumann, Wm. Henry .....Sheboygan, Wis.
Odeneal, Thomas H. .....Winter Haven, Fla.
Page, John Randolph .....New York, N. Y.
Risdon, Fulton .....Toronto, Ont., Canada
Togus, L. Theodore .....Manchester, N. H.

7. Eight Fellows requested that they be allowed to continue under the status of dues remitted. Five new requests were received.

9. The motion picture, Embryology of the Eye, produced by Sturgis-Grant Productions, Inc., aided by Dr. George W. Corner and Dr. George K. Smelser, and sponsored by the Academy, will be exhibited for the first time on Tuesday, October 10. This is the first professionally produced motion picture sponsored by the Academy and sets a standard for teaching films in the basic medical sciences related to ophthalmology and otolaryngology. A suitable plaque has been prepared for each of the principals in the technical production of the film expressing the gratitude of the Academy for their splendid services.

10. At the request of the secretary of the American Ophthalmological Society, Dr. Maynard C. Wheeler, a committee of three was

appointed by President Brown to join similar committees of the American Ophthalmological Society and the Section on Ophthalmology of the American Medical Association in soliciting the next meeting of the International Congress of Ophthalmology for the United States. Appointed on July 5 were Dr. Philip M. Lewis, Memphis, Tenn.; Dr. Erling Hansen, Minneapolis, Minn., and Dr. Bernard Samuels, New York. A report has not been received from this committee.

11. At the request of the secretary of the International Organization Against Trachoma, a representative from the Academy was appointed by President Brown on July 5. Dr. R. Townley Paton of New York was appointed and has submitted his report to the Council through the Activities Committee.

It was moved and seconded that the report of the Executive Secretary be accepted as read. The motion carried.

II

The Treasurer, Dr. Benedict, submitted the audit for 1949, made by Byers, Wobschall & Miller, Certified Public Accountants, and the financial status of the Academy as of Sept. 30, 1950. It was moved and seconded that the report be accepted. The motion carried. (*The audit for* 1949 *appeared in the May-June* 1950 *issue of the* TRANSACTIONS. *The financial status of the Academy as of Sept.* 30, 1950, *is published herewith.*)

EXHIBIT "A"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Balance Sheet as of September 30, 1950

ASSETS

Current Assets:		
Cash, First National Bank, Rochester	$ 42,354.05	
Cash, Bank of Montreal	5,726.71	
Cash, First National Bank, Minneapolis	22,850.67	
Checks for Collection	23.50	
Petty Cash Fund	25.00	
Membership Dues Receivable	470.00	
Motion Picture—Embryology Project	26,200.00	
Inventory—Abstracts & Manuals	11,937.86	
Total Current Assets		$109,587.79
Investments:		
Research Fund	231,738.48	
Wherry Memorial Fund	4,600.00	
	236,338.48	
Less: Reserve Mkt. Fluctuations	2,390.53	
Total Investments		233,947.95
Fixed Assets:		
Furniture & Fixtures	6,623.35	
Less: Reserve for Depreciation	3,344.74	
Total Fixed Assets		3,278.61
Prepaid Insurance		273.87
Prepaid Postage		21.88
Total Assets		$347,110.10

LIABILITIES		
Current Liabilities:		
Accrued Withholding Taxes		$ 205.80
Deferred Income:		
Membership Dues		215.00
Net Worth:		
Balance, January 1, 1950	$278,916.22	
Excess Income over Expenses, Year to Date	67,773.08	
Total		346,689.30
Total Liabilities		$347,110.10

EXHIBIT "B"

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Statement of Income and Expenses for Month Ending September 30, 1950, and Year to Date

	September 1950	1/1/50 to 9/30/50
Revenues:		
Membership Dues	$ 99.00	$ 26,980.00
Membership Applications	225.00	3,936.00
Abstracts & Manuals	976.00	3,173.55
Transactions	2,461.31	34,549.72
Home Study Courses	530.00	3,390.00
Convention	11,576.75	47,189.75
Committee on Standardization of Tonometers	10.00	650.00
Other Income	———	154.84
Income from Securities:		
Research Fund:		
Interest	262.50	6,000.00
Dividends	90.00	937.74
Wherry Memorial Fund:		
Interest	7.50	115.00
Sub-Committee on Noise in Industry	———	4,200.69
Total Revenues	$16,238.06	$131,277.29
Operating Expenses:		
General Administrative	$ 1,954.26	$ 24,617.51
Transactions	2,275.16	24,582.22
Home Study Courses	1,094.41	5,382.46
Research		1,012.50
Convention and Meetings	984.24	7,909.52
Total Operating Expenses	$ 6,308.07	$ 63,504.21
Excess Income over Expenses	$ 9,929.99	$ 67,773.08

American Academy of Ophthalmology and Otolaryngology

Securities Owned as of September 30, 1950

RESEARCH FUND:

BONDS	RATE	MATURITY	PAR VALUE	COST
Canada, Dominion of—Series P5	3	10/ 1/63	$ 2,500.00	$ 2,500.00
Citizens Bldg. Co. of Cleveland, 1st Mortgage	6	1/ 1/46	5,000.00	5,000.00
U.S. Savings Bonds—Series F	2½	12/ 1/55	13,500.00	9,990.00
U.S. Savings Bonds—Series G	2½	8/ 1/53	10,000.00	10,000.00
U.S. Savings Bonds—Series G	2½	6/ 1/55	4,000.00	4,000.00
U.S. Savings Bonds—Series G	2½	3/ 1/56	6,000.00	6,000.00
U.S. Savings Bonds—Series G	2½	5/ 1/56	8,000.00	8,000.00
U.S. Savings Bonds—Series G	2½	7/ 1/56	2,000.00	2,000.00
U.S. Savings Bonds—Series G	2½	11/ 1/56	25,000.00	25,000.00
U.S. Savings Bonds—Series G	2½	1/ 1/57	2,000.00	2,000.00
U.S. Savings Bonds—Series G	2½	7/ 1/58	30,000.00	30,000.00
U.S. Savings Bonds—Series G	2½	12/ 1/58	25,000.00	25,000.00
U.S. Savings Bonds—Series G	2½	3/ 1/59	15,000.00	15,000.00
(6) U.S. Savings Bonds—Series G $5,000.00	2½	7/ 1/60	30,000.00	30,000.00
U.S. Treasury Bond	2½	12/15/72	10,000.00	10,000.00
U.S. Treasury Bond	2½	12/15/72	15,000.00	15,431.25
Van Sweringen Co., Cert. of Indebtedness	6	12/31/48	3,445.70	3,477.03
Total Bonds				$203,398.28

STOCKS	KIND	SHARES	
Citizens Bldg. Co. of Cleveland		50	None
Commonwealth Edison Company	Common	100	2,619.00
DuPont de Nemours & Co.	Common	15	2,347.31
Louisville Gas & Electric Co.	5% Preferred	200	5,450.00
Massachusetts Investors Trust		617	17,923.89
Ophthalmic Publishing Co.	Capital	5	None
Total Stocks			$ 28,340.20
Total		(A)	$231,738.48
WHERRY MEMORIAL FUND: U.S. Savings Bonds—Series G		(B)	$ 4,600.00
			$236,338.48

(A) Held for safekeeping by Trust Dept., First National Bank, Minneapolis, Minn.
(B) Held by Dr. W. L. Benedict, Executive Secretary-Treasurer, Rochester, Minn.

III

The report of the Editor of the TRANSACTIONS was read by Dr. Benedict, Editor-in-Chief, as follows:

For the period September 1, 1949, through August 31, 1950, in addition to the six bimonthly issues of the TRANSACTIONS, a supplement containing papers read at the Otosclerosis Study Group meeting in October 1948 was printed and sent out with a regular mailing. A similar supplement containing papers from the Study Group's 1949 meeting will appear soon.

The Academy has also published four manuals and one monograph during the past year. The Syllabus of Audiometric Procedures in the Administration of a Program for the Conservation of Hearing of School Children has been reprinted. One hundred and forty-one abstracts have been prepared for the 1950 Instruction Section. A manual by Richard Scobee and a monograph by Clarence A. Veasey, Jr., are now being processed.

MANUALS

October 1949—*Woods,* Endogenous Uveitis
March 1950—*Leinfelder,* Neuro-ophthalmology
June 1950—*Berens and Loutfallah,* Ocular Surgery
June 1950—*Sugar,* Extrinsic Eye Muscles (revised edition)

MONOGRAPH

January 1950 —*Juers,* Hearing Tests

In the coming year we will continue to issue a supplement of the papers read at the Special Scientific Program of the Otosclerosis Study Group and will also publish a supplement containing papers read at the Special Scientific Program of the American Society of Ophthalmologic and Otolaryngologic Allergy. Papers read at the meeting of the Committee on Conservation of Hearing (Sunday night meeting) will appear in one of the regular issues of the TRANSACTIONS. Most of the papers read before the Industrial Ophthalmology group will also appear throughout the year in the Industrial Ophthalmology Section of the TRANSACTIONS.

An average of 6092 copies of the TRANSACTIONS was printed bimonthly. Of these, 675 were set aside for bound volumes and an average of 5373 was mailed, an increase in circulation of 348 over last year. The following categories are represented:

Members	4554
Candidates	146
Subscribers	411
Libraries	103
Exchange & Miscellaneous	78
Advertisers	52
Complimentary	29

Four hundred and ninety-five orders for the 1949-1950 bound volume have been received to date.

The average number of pages of paid advertising was 18.7, representing 29 advertisers.

The *revenue* from the TRANSACTIONS was as follows:

Amount set aside from Fellows' dues	$19,844.00
Amount set aside from Candidates' fees	1,202.00
Subscribers (nonmembers)	3,272.00
Bound Volumes	3,018.00
Single issues sold	55.00
Reprints	495.00
Advertisers	9,637.66
Cuts (loaned)	87.55
Total	$37,611.96

Expenditures were as follows:

Printing bimonthly issues	$17,704.74
Binding Bound Volumes	1,097.25
Cuts and engravings	1,664.70
Editor's honorarium	2,000.00
Postage	423.34
Reprints	1,518.92
Directory	3,019.92
Salaries	4,796.00
Supplies (stock, envelopes, etc.)	5,327.73
Total	$37,552.60
Balance	$ 59.36

A motion to accept the report as read was seconded and carried.

IV

Communications

1. The secretary read a communication from Dr. James H. Allen of New Orleans regarding the exemption of medical students, interns and residents in their final years of training. Dr. Derrick Vail moved that the Academy appoint a committee of ex-medical officers who had served in World War II to be known as the Committee on Armed Forces. The motion was seconded by Dr. Erling Hansen and carried. The following committee was appointed: Dr. James N. Greear, Jr., Washington, D. C., chairman; Dr. Brittain F. Payne, New York; Dr. Gordon D. Hoople, Syracuse, N. Y.; Dr. Gordon M. Bruce, New York; and Dr. Harry P. Schenck, Philadelphia. The committee is to formulate opinions of policy regarding exemptions and will represent the Academy as a liaison committee with the Armed Forces.

2. Dr. A. F. MacCallan of London wrote to Dr. R. Townley Paton in regard to the London activities of the International Organization Against Trachoma and asked for a donation from the Academy to assist this organization in its work. Dr. Paton, who represented the Academy at the meeting of the organization in June of this year, referred the letter to the Executive Secretary. The Council voted to send $25.00 to the organization.

3. The National Society for Crippled Children and Adults, Inc., requested "the official action of the American Academy of Ophthalmology and Otolaryngology to create a formal liaison relationship with the National Society for Crippled Children and Adults." The Council approved the request, and Dr. Dean M. Lierle was appointed to the position of liaison officer.

4. The Secretary presented a letter written by Captain W. L. Berkley, MC, USN, concerning the Sixth Brazilian Jornadas of Ophthalmology, which took place in Sao Paulo, Brazil, September 3-7, 1950. Dr. Berkley participated in the program. He praised the excellence of the scientific program and the manifestations of the very warm friendship which the people of Brazil have toward the United States. He commented on the importance of a Good Neighbor policy and urged more participation in the ophthalmologic meetings of Central and South American countries.

5. A communication addressed to the President and signed by Dr. C. E. Kinney, concerning the advertising of cigarette manufacturers, was read to the Council. There was no specific recommendation and, after some discussion, the matter was laid on the table for further consideration.

6. A communication was submitted by Dr. Hedwig Kuhn as secretary of the Joint Committee on Industrial Ophthalmology urging the approval or disapproval of the recommendations of the 1949 meeting of the Joint Committee relative to monocular aphakia and compensation. Inasmuch as matters of compensation are handled by the American Medical Association, the Council recommended that the matter be referred to the Association.

V

Report of the Executive, Research and Finance Committee—Dr. J. Mackenzie Brown, chairman.

1. The Committee recommends that the application fee shall be $25.00, and that the annual dues for Junior Fellows shall continue to be $12.00, and for Senior Fellows, $6.00. The allotment from dues for subscriptions to the TRANSACTIONS shall remain at $5.00. It was moved and seconded that this recommendation be approved. The motion carried.

2. The Committee recommends that the 10 resignations of Fellows from the Academy reported by the Executive Secretary be accepted. A motion to this effect was seconded and carried.

4. The Committee recommends that Dr. George W. Corner, Baltimore, Md., and Dr. George K. Smelser, New York City, be made Honorary Fellows of the Academy. This recommendation was approved.

5. The Committee recommends the following appointments to Standing and Joint Committees:

To the Advisory Committee to the Registries of Pathology, Dr. Brittain F. Payne, chairman.

To the Committee on Conservation of Hearing, Dr. Dean M. Lierle, chairman.

To the Committee on Pan-American Relations in Otolaryngology, Dr. C. L. Jackson, chairman; Dr. Norton Canfield; Dr. A. C. Furstenberg; Dr. Gordon B. New; Dr. Theodore E. Walsh; and Dr. Paul H. Holinger. At the request of Dr. Jackson, Dr. Victor Alfaro of Washington, D. C., was added to the Committee.

To the Committee on Prevention of Blindness, Dr. Ralph I. Lloyd, chairman; Dr. Lawrence T. Post; and Dr. Alan C. Woods.

To the Committee on Scientific Exhibit, Dr. Kenneth L. Roper, chairman.

To the Committee on Standardization of Tonometers, Dr. Jonas S. Friedenwald, chairman.

To the Committee on the Study of Audio-Visual Instruction, Dr. Dean M. Lierle, chairman; Dr. W. L. Benedict; Dr. Howard P. House, Dr. Algernon B. Reese; and Dr. A. D. Ruedemann.

To the Advisory Committee to the Eye Health Committee of the American Student Health Association, Dr. Lawrence T. Post.

To the American Association for the Advancement of Science, Dr. M. Elliott Randolph.

To the American Board of Ophthalmology, Dr. F. Bruce Fralick.

To the American Board of Otolaryngology, Dr. C. H. McCaskey and Dr. W. E. Grove.

To the American College of Surgeons Board of Governors, Dr. James H. Maxwell.

To the American Committee on Optics and Visual Physiology, Dr. Alfred Cowan.

To the American Orthoptic Council, Dr. Kenneth C. Swan.

To the American Society of Clinical Pathologists Consultative Panel on Tumor Terminology, Dr. John S. McGavic.

To the Joint Committee on Industrial Ophthalmology, Dr. Edmund B. Spaeth, Dr. John B. Hitz, and Col. Victor A. Byrnes (MC).

It was moved and seconded that these appointments be approved. The motion carried.

6. The Committee recommends that the Helmholtz Centenary Committee be discharged with thanks for the services rendered. The recommendation was approved.

7. The Committee nominates Dr. William L. Benedict as Editor-in-Chief of the TRANSACTIONS. It was moved and seconded that the nomination be approved. The motion carried.

8. The Committee recommends that the next annual meeting of the Academy be held October 14-19, 1951, at the Palmer House, Chicago. The recommendation was approved. The Council directed the Executive Secretary-Treasurer to arrange for future meetings of the Academy.

9. The Committee recommnds the following appropriations for the calendar year 1951:

To the Advisory Committee to the Registries of Pathology, a sum not to exceed $8,500 toward the completion of the atlases. This is to include the services of a fellow in ophthalmology and one in otolaryngology.

To the Committee on Conservation of Hearing, a sum not to exceed $9,000.

To the Committee on Standardization of Tonometers, a sum not to exceed $1,000.

To the American Committee on Optics and Visual Physiology, $100.

To the American Orthoptic Council, $200 if requested.

To the Joint Committee on Industrial Ophthalmology, a sum not to exceed $1,000, if matched by an equal sum from the American Medical Association and if the Academy is accorded proper credit for its participation in this Committee.

To the National Research Council, $2,500.

To the National Society for Medical Research, $1,000.

On the motion of Dr. Algernon Reese, seconded by Dr. Erling Hansen, the report of the Committee was accepted and the recommendations adopted.

VI

Report of the Activities Committee—Dr. Thomas D. Allen, chairman.

Reports of the following committees were reviewed and approved by the Activities Committee:

1. Advisory Committee to the Registries of Pathology
2. Committee on Conservation of Hearing
3. Committee on Scientific Exhibit
4. Committee on Standardization of Tonometers
5. Committee on the Prevention of Blindness
6. Committee on the Study of Audio-Visual Instruction
7. Advisory Committee to the Eye Health Committee of the American Student Health Association
8. American Association for the Advancement of Science
9. American Board of Ophthalmology
10. American Board of Otolaryngology
11. American Committee on Optics and Visual Physiology
12. American Orthoptic Council
13. Helmholtz Centenary Committee
14. Joint Committee on Industrial Ophthalmology
15. American Society of Clinical Pathologists Consultative Panel on Tumor Terminology
16. Representative to the International Organization Against Trachoma

(*Full reports of standing and joint committees follow the report of the Business Meeting.*)

The report of the Committee on Pan-American Relations in Otolaryngology was given verbally by the chairman, Dr. C. L. Jackson, who was instructed to submit a written report to the Chairman of the Activities Committee before the meeting of the Council on Wednesday.

No reports were received from:

1. American College of Surgeons Board of Governors
2. Committee on Meeting of the International Congress of Ophthalmology

A motion was made and seconded that the report of the Activities Committee be accepted as a whole. The motion carried.

VII

Report of the Board of Councillors—Dr. Thomas D. Allen, chairman.

1. Nomination of officers:

President—Dr. Derrick Vail, Chicago, Ill.
President-Elect—Dr. James Milton Robb, Detroit, Mich.
First Vice-President—Dr. Francis E. LeJeune, New Orleans, La.
Second Vice-President—Dr. Peter Kronfeld, Chicago, Ill.
Third Vice-President—Dr. Frederick A. Figi, Rochester, Minn.
Councillor—Dr. John H. Dunnington, New York, N. Y.
Executive Secretary-Treasurer—Dr. William L. Benedict, Rochester, Minn.
Secretary for Ophthalmology—Dr. Algernon B. Reese, New York, N. Y.
Secretary for Otolaryngology—Dr. Howard House, Los Angeles, Calif.
Secretary for Instruction in Ophthalmology—Dr. A. D. Ruedemann, Detroit, Mich.
Secretary for Instruction in Otolaryngology and Maxillofacial Surgery—Dr. Dean M. Lierle, Iowa City, Iowa.

Secretary for Home Study Courses—Dr. Lawrence R. Boies, Minneapolis, Minn.
Secretary for Public Relations—Dr. Erling W. Hansen, Minneapolis, Minn.

It was moved and seconded that the recommendations of the Board of Councillors for the officers of the Academy for 1951 be accepted. The motion carried.

3. The Board of Councillors recommends that 285 candidates be accepted for Fellowship in the Academy:

(*The names of candidates for fellowship approved by the Council are published in the account of the Business Meeting.*)

A motion to accept these recommendations was seconded and carried.

VIII

Report of the Judicial Committee—Dr. J. Mackenzie Brown, chairman.

There was no business for the Judicial Committee and a meeting of the Committee was not required.

IX

New Business

1. It was moved by Dr. Alan Woods and seconded by Dr. W. L. Benedict that the following addition be made to Article V, Section 6, of the By-Laws:

(Following identification of Junior Fellows)

Inactive Fellows: Senior or Junior Fellows who by reason of physical infirmity or retirement from practice may by action of the Council be classified as "Inactive." Inactive Fellows shall pay no dues, have no voting privileges, and shall not receive the TRANSACTIONS.

The motion carried.

2. Dr. Alan Woods moved that the research fund be renamed Educational Fund. The motion was seconded by Dr. W. L. Benedict and carried.

3. At the January meeting of the Board of Secretaries, the organization of a Teachers' Section was recommended. It was moved by Dr. A. D. Ruedemann and seconded by Dr. Erling Hansen that the Council create a Teachers' Section as an Academy function. The motion carried and Dr. Lawrence R. Boies was named chairman and given the privilege of adding to his Committee by appointment.

4. The management of a special fund to be known as "The Isabella Wilson Research and Educational Fund for Frontal Sinus Pathology," initiated by Dr. Robert H. Fraser of Battle Creek, Mich., was accepted by the Council. The purpose of this fund was set forth in a letter addressed to the Secretary under date of October 9, 1950. This is tentatively a fund of $500, $250 contributed from the estate of Miss Isabella Wilson, and contributions of not over $10.00 to be accepted from interested physicians and others. A check for $150 signed by Dr. Robert H. Fraser accompanied the letter.

5. The secretary of the American Orthoptic Council, Dr. Frank J. Costenbader, in a communication to the Secretary requested that the Academy publish certain papers from the Special Scientific Program of the American Association of Orthoptic Technicians and the American Orthoptic Council. It is the hope of the American Association of Orthoptic Technicians and the American Orthoptic Council that additional material may be combined with the scientific papers to make up a publication to be known as the *American Orthoptic Journal*. A motion was made by Dr. Alan Woods and seconded by Dr. Conrad Berens that the proposal be accepted, the arrangements to be completed by Dr. Derrick Vail and Dr. W. L. Benedict. The motion carried.

6. Dr. Thomas D. Allen moved that the Academy have an exhibit at the Meeting of the Pan-American Congress of Ophthalmology in Mexico City in 1952. It was suggested that the Academy and the American Board of Ophthalmology might plan a joint exhibit. The motion was seconded by Dr. Alan Woods and carried. The Council authorized the expenses of Miss Maud Givens to accompany the exhibit.

8. The Joint Committee on Industrial Ophthalmology presented for endorsement by the Council the following recommendations of basic principles, objectives and essentials to be met by manufacturer, distributor or any other representative, for introducing an effective and ethical visual testing program to industry:

1. The manufacturer, distributor or any other representative to recommend only acceptable and proper instrumentation for the testing of visual skills.
2. The presentation of currently acceptable visual testing procedures shall be made to the medical director and/or professional eye consultant of plant by the salesmen.
3. Insistence in sales approach that interpretation, evaluation of records and all referral and correction programs are the re-

sponsibility of the medical director and/or professional eye consultant.

4. The presentation of the over-all subject of instrument testing of visual skills in industry shall be made to local professional groups and societies whenever at all possible.
5. At no time shall a salesman of such instrumentation indicate to lay personnel of industry that professional guidance is not necessary.
6. Any job standards and/or "profiles" presented to the industry must be based on research available both to the industry purchasing the instrument and/or the professional eye consultant.
7. When an instrument as such is sold outright to the company without an additional continuous service, the price of the instrument should be for the instrument alone. It would be desirable that instrumentation be sold outright to plants that have professional eye consultants.
8. The attitude and bearing of any salesman asking for an interview with a professional individual shall be in line with accepted ethical standards.

A motion to endorse these recommendations was made by Dr. A. D. Ruedemann, seconded by Dr. Derrick Vail, and carried.

The meeting adjourned.

ANNUAL BUSINESS MEETING

The business meeting of the Fifty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology convened at 5:30 p.m. in the Crystal Room of the Palmer House, Chicago, Oct. 12, 1950. President J. Mackenzie Brown presided.

Report of the Executive Secretary-Treasurer, Dr. William L. Benedict

The fellowship in this Academy as of Oct. 1, 1950: Junior Fellows, 3,907; Senior Fellows, 150; Life Fellows, 427; Honorary Fellows, 26; making a total of 4,510. Candidates to be considered at this meeting, 285.

Elevation to senior fellowship on Jan. 1, 1951, 41.

Elevation to life fellowship on Jan. 1, 1951, 17.

Deaths reported since the last meeting, 53.

Resignations received, 10.

[*See* Council Minutes.]

Review of those whose dues have been remitted and who are carried on the membership roll at their request, 13.

On motion of Dr. Alan C. Woods, seconded by Dr. John J. Shea, the report was accepted as read.

Report of the Senior Member of the Council, Dr. Alan C. Woods

Mr. President and Members: The Council has received and approved the reports of the Treasurer, the Editor of the TRANSACTIONS, the Council Committees, and joint and standing committees. These reports will be published in the November-December issue of the TRANSACTIONS. [*See* Council Minutes *and* Committee Reports.]

DR. WOODS: The Council recommends that the application fee for fellowship in the Academy shall remain at $25.00, and that annual dues for Junior Fellows shall be $12.00; for Senior Fellows, $6.00.

A motion to accept the recommendation of the Council was made by Dr. Carl H. McCaskey and seconded by Dr. James M. Robb. The motion carried.

DR. WOODS: A request for financial assistance to support the International Organization Against Trachoma was directed to the Academy by Dr. A. F. MacCallan, secretary. The Council recommends the appropriation of $25.00 to the organization.

On motion of Dr. Thomas Allen, seconded by Dr. Shea, the recommendation of the Council was accepted.

DR. WOODS: The Council recommends the following appropriations for the year 1951:

To the Advisory Committee to the Registries of Pathology, a sum not to exceed $8,500 toward the completion of the atlases. This is to include the services of a fellow in ophthalmology and one in otolaryngology at the Armed Forces Institute of Pathology in Washington, D. C.

To the Committee on Conservation of Hearing, a sum not to exceed $9,000.

To the Committee on Standardization of Tonometers, a sum not to exceed $1,000.

To the American Committee on Optics and Visual Physiology, $100.

To the American Orthoptic Council, $200 if requested.

To the Joint Committee on Industrial Ophthalmology, a sum not to exceed $1,000, if matched by an equal sum from the American Medical Association.

To the National Research Council, $2,500.

To the National Society for Medical Research, $1,000.

On motion by Dr. McCaskey, seconded by Dr. Allen, the appropriations recommended by the Council were accepted.

DR. WOODS: The Council recommends the following appointments to standing and joint committees for the year 1951:

To the Advisory Committee to the Registries of Pathology, Dr. Brittain F. Payne, chairman.

To the Committee on Conservation of Hearing, Dr. Dean M. Lierle, chairman.

To the Committee on Pan-American Relations in Otolaryngology, Dr. C. L. Jackson, chairman; Dr. Norton Canfield; Dr. A. C. Furstenberg; Dr. Gordon B. New; Dr. Theodore E. Walsh; Dr. Paul H. Holinger; and Dr. Victor Alfaro.

To the Committee on Prevention of Blindness, Dr. Ralph I. Lloyd, chairman; Dr. Lawrence T. Post; and Dr. Alan C. Woods.

To the Committee on Scientific Exhibit, Dr. Kenneth L. Roper, chairman.

To the Committee on Standardization of Tonometers, Dr. Jonas S. Friedenwald, chairman.

To the Committee on the Study of Audio-Visual Instruction, Dr. Dean M. Lierle, chairman; Dr. W. L. Benedict; Dr. Howard P. House; Dr. Algernon B. Reese; and Dr. A. D. Ruedemann.

To the Advisory Committee to the Eye Health Committee of the American Student Health Association, Dr. Lawrence T. Post.

To the American Association for the Advancement of Science, Dr. M. Elliott Randolph.

To the American Board of Ophthalmology, Dr. F. Bruce Fralick.

To the American Board of Otolaryngology, Dr. C. H. McCaskey and Dr. W. E. Grove.

To the American College of Surgeons Board of Governors, Dr. James H. Maxwell.

To the American Committee on Optics and Visual Physiology, Dr. Alfred Cowan.

To the American Orthoptic Council, Dr. Kenneth C. Swan.

To the American Society of Clinical Pathologists Consultative Panel on Tumor Terminology, Dr. John S. McGavic.

To the Joint Committee on Industrial Ophthalmology, Dr. Edmund B. Spaeth, Dr. John B. Hitz, and Col. Victor A. Byrnes (MC).

On motion by Dr. McCaskey, seconded by Dr. Allen, the appointments recommended by the Council were accepted.

DR. WOODS: The Council was requested to give suggestions and advice regarding the exemption of medical students and residents from military service. A committee to be known as "The Committee on Armed Forces" was appointed by the President. The Committee consists of Dr. James N. Greear, Jr., chairman; Dr. Brittain F. Payne; Dr. Gordon D. Hoople; Dr. Gordon M. Bruce; and Dr. Harry P. Schenck.

On motion by Dr. Shea, seconded by Dr. Derrick Vail, the action of the Council was approved.

DR. WOODS: The National Society for Crippled Children and Adults, Inc., has requested official action of the Academy to create a formal liaison relationship with the Society. The Council recommends the appointment of Dr. Dean M. Lierle as liaison officer.

On motion of Dr. A. D. Ruedemann, seconded by Dr. Shea, the recommendation of the Council was accepted.

DR. WOODS: The Council recommends the organization of a Teachers' Section. The purpose of this section would be to set up a forum for discussion of problems pertinent to teaching of the specialties in medicine. The Council has recommended that Dr. Lawrence R. Boies be named chairman and be given the privilege of adding to his committee by appointment.

Upon motion by Dr. Vail, seconded by Dr. Allen, the recommendations of the Council were accepted.

DR. WOODS: The Council nominates for honorary fellowship in the Academy, Dr. George W. Corner and Dr. George K. Smelser. The Council also nominates for active fellowship the 285 candidates whose names appear on the sheets handed you when you came in the door.

On motion by Dr. Carroll Mullen, seconded by Dr. Allen, the nominations made by the Council were accepted.

Newly elected Fellows of the Academy:

Acquarelli, Mario John, Wadsworth General Hospital, Los Angeles 25, Calif., ALR.

Albers, George Donald, 8½ Monroe St., Grand Rapids 2, Mich., ALR.

Anderson, Elam DeMar, Medical & Dental Bldg., Seattle 1, Wash., OP.

Anderson, Elbert Carl, 201 N. Front St., Wilmington, N. C., OP.

Anderson, H. Gordon, 403 Fulton St., Troy, N. Y., OP.

Anslow, Robert Elmer, 10 Peterboro St., Detroit 1, Mich., OP.

Baers, Harry Arnold, 4063 Radford Ave., Studio City, Calif., ALR.

Baldridge, Max N., P.O. Box 778, Texarkana, Ark.-Texas, OP.

Barbee, John Young, 1109 State St., Bowling Green, Ky., ALR.

Barrere, Luciano Enrique, Tulane University, New Orleans 12, La., OP.

Barton, William Lawrence, 403 Persons Bldg., Macon, Ga., ALR.

Beasley, Clifton Harold, 1216 Pennsylvania Ave., Fort Worth, Texas, OP.

Bergman, Macks Leonard, 8443 Crenshaw Blvd., Inglewood, Calif., ALR.

Birmingham, Eugene E., 333 W. North Ave., Chicago 10, Ill., ALR.

Blair, James Robert, 920 Metropolitan Bldg., Denver 2, Colo., ALR.

Blanford, Sidney Edgar, Jr., 1624 Gilpin St., Denver, Colo., Pl S.

Blodi, Frederick C., 635 W. 165th St., New York 32, N. Y., OP.

Bloomberg, Louis, 506 Central Tower Bldg., Youngstown 3, Ohio, OP.

Bobbett, Gordon Howard, 125 W. Cheves St., Florence, S. C., ALR.

Boshnack, Malcolm, 70 Strawberry Hill Ave., Stamford, Conn., ALR.

Bosworth, Wesley F., E. Main St., Clarinda, Iowa, OP.

Boucher, Irvan Andrew, 1221 12th Ave., Altoona, Pa., ALR.

Braveman, Bernard Leon, 412 Fifth Ave., McKeesport, Pa., OP.

Breffeilh, Louis Andrew, 421 Medical Arts Bldg., Shreveport, La., OP.

Brown, Kenneth Brien, 131 Fulton Ave., Hempstead, N. Y., ALR.

Bryan, John Thomas, 2105 Hayes St., Nashville 5, Tenn., ALR.

Burnham, Charles Joseph, 1529 N. 25th St., Birmingham, Ala., OP.

Burr, Sherwood Petersen, 43 E. Jackson St., Tucson, Ariz., OP.

Butler, Jay B. V., 919 Taylor Street Bldg., Portland 5, Ore., OP.

Cairns, Adrian Bennett, 415 Pere Marquette Bldg., New Orleans 12, La., ALR.

Callaghan, Winship C., Union Trust Bldg., Greensburg, Ind., ALR.

Callahan, Neil, 315 Medical Arts Bldg., Norfolk 10, Va., ALR.

Cammack, Bragg Charles, 6381 Hollywood Blvd., Hollywood 28, Calif., ALR.

Capriotti, Octavius A., 404 E. Broad St., Souderton, Pa., OP.

Carris, James Vernon, 209 S. Nevada Ave., Colorado Springs, Colo., ALR.

Casey, Edwin Joseph, 508 N. Grand Ave., St. Louis 3, Mo., OP.

Christenberry, Kenneth William, 501 W. Church Ave., Knoxville, Tenn., OP.

Clark, Archibald Fletcher, Jr., 225 Medical Arts Bldg., San Antonio 5, Texas, ALR.

Clay, Richard A., 416 N.W. 13th St., Oklahoma City, Okla., OP.

Clayton, Sam, 119-05 80th Road, Kew Gardens 15, N. Y., ALR.

Coleman, Howe Reese, 305 Young Bldg., Lynchburg, Va., ALR.

Corgill, Donald Alton, Veterans Administration Hospital, McKinney, Texas, ALR.

Crane, Edward Harrison, Jr., 127 S. Grevillea Ave., Inglewood, Calif., ALR.

Crawford, Walter James, 3333 Pachappa Drive, Riverside, Calif., OP.

Cressman, Frederic E., 102 S. Second St., Artesia, N. M., OP.

Crowder, Miles S., 603 W. Main St., Knoxville, Tenn., OP.

Daly, Joseph M., 1801 K St. N.W., Washington 6, D. C., ALR.

DeLuca, Charles Q., 255 S. 17th St., Philadelphia 3, Pa., ALR.

Denicke, Ernest Webber, 1010 B St., San Rafael, Calif., OP.

Dennis, Richard Hollis, 33 College Ave., Waterville, Maine, OP.

Dillahunt, Jack A., Copper Ave. at Monroe, Albuquerque, N. M., OP.

Dorman, Purman, 1215 Fourth Ave., Seattle 1, Wash., OALR.

Dryden, James Spencer, 1835 Eye St. N.W., Washington 6, D. C., OP.

Duncan, Herbert, 700 Church St., Nashville 3, Tenn., ALR.

duPrey, Robert E., 1150 Connecticut Ave., Washington 6, D. C., OP.
Dwyer, Gregory Kennedy, Community Medical Group, Boonton, N. J., ALR.

Edelstein, Isidore S., 259 New York Ave., Brooklyn 16, N. Y., OP.
Edwards, Thomas Luther, 507 S. Washington St., Van Wert, Ohio, OP.
Egdorf, Otto Charles, 420 Hamilton Bldg., Wichita Falls, Texas, ALR.
Esbin, Leo, 1333 President St., Brooklyn 13, N. Y., OP.
Esposito, Albert Charles, First Huntington National Bank Bldg., Huntington 1, W. Va., OP.
Eubank, William Richards, 1102 Grand Ave., Kansas City 6, Mo., OP.
Evans, John W., 325 Franklin St., Huntsville, Ala., ALR.

Failla, Anthony, 3831 Frenchmen St., New Orleans, La., ALR.
Fairbanks, Stephen, 119½ N. Superior St., Albion, Mich., OP.
Feher, Alexander, 123 E. 83rd St., New York 28, N. Y., ALR.
Feldman, John L., 416 S. 24th St., Quincy, Ill., ALR.
Feldstein, Morris, 515 Park Ave., New York 22, N. Y., OP.
Fields, James Allan, Naval Hospital, Beaufort, S. C., ALR.
Florentz, Theodore Robert, 109 N. Eighth St., Boise, Idaho, OP.
Follette, William James, 375 Engle St., Englewood, N. J., ALR.
Freeman, David M., McMillan Hospital, St. Louis 12, Mo., OP.
Friedman, Isadore Edward, 5248 Hohman St., Hammond, Ind., ALR.
Friedman, Paul Norman, 1111 St. Paul St., Baltimore 2, Md., OP.
Fuchs, Jesse, 6363 Wilshire Blvd., Los Angeles 48, Calif., ALR.

Gardner, James Frank, 11 N. Goodman St., Rochester, N. Y., ALR.
Garner, Lawrence Lee, 238 W. Wisconsin Ave., Milwaukee 3, Wis., OP.
Garron, Levon K., 426 17th St., Oakland 12, Calif., OP.
Gifford, Edward Stewart, Jr., 1913 Spruce St., Philadelphia 3, Pa., OP.
Gillman, A. Marvin, 54 E. 72nd St., New York 21, N. Y., OP.
Girard, Louis Joseph, Centre Island, Long Island, N. Y., OP.
Glass, Walter Martin, 221 Middle Neck Rd., Great Neck, N. Y., ALR.
Godwin, Robert William, 117 E. Eighth St., Long Beach 13, Calif., ALR.
Golden, Samuel Charles, 150 Prichard St., Fitchburg, Mass., ALR.
Goldsmith, Charles Porter, 1648 Hamilton St., Allentown, Pa., OP.
Gooch, J. Oliver, 1203 Hermann Professional Bldg., Houston 5, Texas, ALR.
Gorin, George, 885 West End Ave., New York 25, N. Y., OP.
Grayman, Harry Myer, 2900 Fresno Street Bldg., Fresno 1, Calif., OP.
Greene, Richard W., 32 Johnson Ave., Newark 8, N. J., OP.
Greenfield, S. Jerome, 31 Lincoln Park, Newark 2, N. J., OP.
Greer, Rex E., 203 Fisk Bldg., Amarillo, Texas, ALR.
Gros, Jose Conrad, Calle 25 no. 960, Vedado, La Habana, Cuba, ALR.
Guida, Francis Paul, 67 Trumbull St., New Haven 10, Conn., OP.

Haight, Whitney James, 9 Exchange Place, Salt Lake City 1, Utah, ALR.
Harms, Edwin M., 104 W. Douglas St., Wichita 2, Kan., OP.
Harper, Donald McCarthy, 1915 New Hampshire Ave., N.W., Washington 9, D. C., ALR.
Hatfield, Haskell Doke, 1201 First National Bldg., El Paso, Texas, ALR.
Hathcock, William Caldwell, 402 Grand Theatre Bldg., Atlanta 3, Ga., OP.
Heck, Walter Emil, Stanford University Hospital, San Francisco, Calif., ALR.
Heller, Morris Freund, 115 E. 61st St., New York 21, N. Y., ALR.
Herman, Seymour J., 1601 E. 23rd St., Brooklyn 29, N. Y., ALR.
Hirst, William Randolph, 2241 Central Ave., Alameda, Calif., OP.
Hoch, Carl William, 2904 Wilbur St., Rockford, Ill., ALR.
Hoffman, Franklin David, 626 Union Trust Bldg., Pittsburgh 19, Pa., OP.
Hoffman, Parker M., 134 E. First St., Corning, N. Y., OP.
Hogg, Stephen P., 556 Doctors' Bldg., Cincinnati 2, Ohio, ALR.
Hosner, James Wesley, 255 S. 17th St., Philadelphia 3, Pa., OP.
Hull, Forrest Edgar, Tokyo General Hospital, APO 1052, c/o PM, San Francisco, Calif., OP.
Huston, James M., 1280 E. 14th St., San Leandro, Calif., ALR.
Hynes, Edward Allen, U. S. Naval Academy, Annapolis, Md., OP.

Hyslop, Volney Butman, 759 N. Milwaukee St., Milwaukee 2, Wis., PI S.

Ittkin, Paul, 5921 Clark St., Montreal, Que., Canada, ALR.

Jennings, Edward C., 2650 Wisconsin Ave., Washington 7, D. C., ALR.

Kant, Alfred, 129 Clinton St., Watertown, N. Y., OP.
Katz, Jacob, 1807 S. Sixth St., Philadelphia 48, Pa., OP.
Keck, William Struble, 122 Main St., Greensburg, Pa., ALR.
Kelemen, George, 20 Gloucester St., Boston 15, Mass., ALR.
Kennon, William G., Jr., 706 Church St., Nashville 3, Tenn., ALR.
Kimmelman, David Brown, 10 Downing St., New York 14, N. Y., OP.
Kirsch, Ralph Emile, 903 Huntington Bldg., Miami 32, Fla., OP.
Kirshner, Harold, 20 Park Ave., New York 16, N. Y., OP.
Knapp, Philip, University Hospitals, Iowa City, Iowa, OP.
Kolodny, George Robert, 702 Hermann Professional Bldg., Houston 5, Texas, OP.
Krishna, Ikbal, 1049 E. Washington St., Brownsville, Texas, ALR.
Krug, Joseph Hoffmann, 988 Fifth Ave., New York 21, N. Y., OP.
Kunkel, William Howard, U.S. Army Hospital, Camp Atterbury, Ind., ALR.

Lane, Charles Spurgeon, Jr., 205 Merchants National Bank Bldg., Fort Smith, Ark., ALR.
Lateiner, Robert, 650 Main St., New Rochelle, N. Y., OP.
Latella, Peter D., 524 North Ave., New Rochelle, N. Y., ALR.
Lauren, George Peter, 205 Medico-Dental Bldg., San Diego 1, Calif., OP.
Lavoie, Roland, 3 Claire-Fontaine, Quebec City, Que., Canada, ALR.
Lebo, Charles Phillip, 655 Sutter St., San Francisco 2, Calif., ALR.
Lee, Jack Bennett, 607 New Moore Bldg., San Antonio 5, Texas, OP.
Lewis, Paul M., 519 N. Highland Ave., Pittsburgh 6, Pa., ALR.
Lhotka, Frank M., 6005 Cermak Road, Cicero 50, Ill., OP.
Liebman, Sumner David, 115 Bay State Road, Boston 15, Mass., OP.
Linden, Arthur Joseph, Shirlington Theatre Bldg., Arlington, Va., ALR.
Locklin, Walter Kaye, 1410 American National Bank Bldg., Kalamazoo, Mich., ALR.
Loring, Milton Jack, 304 North 'N' St., Midland, Texas, OP.
Love, William Robert, 321 Main St. E., Hamilton, Ont., Canada, ALR.
Lovely, David K., 73 Deering St., Portland 4, Maine, ALR.
Lowrey, Austin, Walter Reed Hospital, Washington 12, D. C., O.P.
Loy, David Taylor, 3207 W. 16th St., Great Bend, Kan., OP.
Luedde, Fullerton Woods, 35 N. Central Ave., St. Louis (Clayton) 5, Mo., OP.
Lymberis, Marvin Nicholas, 106 W. Seventh St., Charlotte 2, N. C., OP.

MacMillan, Charles Wright, 4 Duryea Road, Upper Montclair, N. J., ALR.
Magnet, Isaac Harry, 130 Rock St., Fall River, Mass., ALR.
Maloney, Walter Hugh, Green and Coulter Sts., Philadelphia 44, Pa., ALR.
Mandelbaum, Joseph, 101 Lafayette Ave., Brooklyn 17, N. Y., OP.
Mannis, Aaron A., 109 N. Wabash Ave., Chicago 2, Ill., OP.
Marshall, Alexander Washington, 145-A Rutledge Ave., Charleston 17, S. C., OP.
Mathes, William Thomas, Jr., Jones EENT Hospital, Johnson City, Tenn., ALR.
McFarland, James Jerry, 1150 Connecticut Ave., N.W., Washington 6, D. C., ALR.
McGee, Hugh E., 117 Cedar Lane, Teaneck, N. J., OP.
McGowan, William Louis, Union Central Bldg., Cincinnati 2, Ohio, OP.
McKeigue, John E., 94 Pleasant St., Arlington, Mass., ALR.
Medof, Milton I., 130 S. Robertson Blvd., Beverly Hills, Calif., ALR.
Merriam, George Rennell, Jr., 635 W. 165th St., New York 32, N. Y., OP.
Merz, Arthur Edgar, 821 Franklin Ave., Garden City, N. Y., OP.
Meyer, Eugene A., 427 Cooper St., Camden 2, N. J., ALR.
Michelson, Robin P., 240 Stockton, San Francisco 8, Calif., ALR.
Mietus, Conrad A., 930 Fillmore Ave., Buffalo 11, N. Y., OP.
Milanese, Nicholas Peter, 123 E. 83rd St., New York 28, N. Y., ALR.
Miller, Jack Barnett, 4212 N.E. Broadway, Portland 13, Ore., ALR.

Miller, William Jacob, 21 E. State St., Columbus, Ohio, ALR.
Minnes, James Fortin, 1701 W. Broadway, Vancouver, B. C., Canada, OP.
Mishler, Jay Eli, 1616 Pacific Ave., Atlantic City, N. J., OP.
Mitchell, Howard Lysle, 103 S. Main St., Lexington, Va., ALR.
Montgomery, Earl Clifton, 1620 Medical Arts Bldg., Omaha 2, Neb., ALR.
Moore, Ralph Lewis, 509 N. Broad St., Woodbury, N. J., ALR.
Moorman, Victor Reuben, Wiley Bldg., Hutchinson, Kan., ALR.
Moreland, Joseph Ivan, 2485 Center St., Salem, Ore., OP.
Morgenstern, David Jacob, 433 Eastern Parkway, Brooklyn 16, N. Y., ALR.
Morrison, Lewis E., 503 Hume Mansur Bldg., Indianapolis 4, Ind., ALR.
Moulton, Everett Crockett, Jr., 205 Merchants National Bank, Fort Smith, Ark., OP.
Mulberger, Robert D., 1930 Chestnut St., Philadelphia 3, Pa., OP.
Myers, Roland Horace, 1720 Exchange Bldg., Memphis 3, Tenn., OP.

Nakashima, Victor Katsuhiro, Veterans Administration, Des Moines 10, Iowa, ALR.
Nickeson, Robert Warren, 179 Allyn St., Hartford 3, Conn., OP.
Nisbet, Alfred Alan, 1110 South Texas Bldg., San Antonio, Texas., OP.
Noble, Bertha Riveroll, 1430 Tulane Ave., New Orleans 12, La., OP.

O'Connell, John D., 50 Farmington Ave., Hartford 5, Conn., OP.
Olson, James Albert, Henry Ford Hospital, Detroit 2, Mich., OP.
O'Neill, John Campbell, 205 W. Second St., Duluth, Minn., OP.
Orzac, Edward Seymour, 460 Rockaway Ave., Valley Stream, N. Y., ALR.

Parker, Francis William, 1102 Broadway, Rockford, Ill., OP.
Parks, Kirtland Garvin, 605 Professional Bldg., Long Beach 13, Calif., OP.
Paul, Thomas Otis, 2205 Highland Ave., Birmingham 5, Ala., OP.
Pedersen, Paul Milton, 2241 Central Ave., Alameda, Calif., ALR.
Pendexter, Sidney Eugene, Jr., 11 S. Arlington Ave., East Orange, N. J., OP.
Perzia, Anthony Peter, 910 Citizens Bldg., Tampa 2, Fla., OP.
Peterson, John Hartley, 812 Medical Arts Bldg., Duluth, Minn., OP.
Płotke, Harry L., 1058 Lowry Medical Arts Bldg., St. Paul 2, Minn., OP.
Pole, Samuel Boyce, III, 401 E. Commerce St., Bridgeton, N. J., OP.
Polisar, Ira Allan, 142 Joralemon St., Brooklyn 2, N. Y., ALR.
Powell, James Robert, 501 Medico-Dental Bldg., Stockton, Calif., OP.
Proctor, Malvin, 66 Main St., Tuckahoe 7, N. Y., OP.

Rackwitz, George, 245 E. Broadway, New York 2, N. Y., OP.
Rambo, John Henry T., 119 E. 74th St., New York 21, N. Y., ALR.
Rea, Robert P., Gorgas Hospital, Ancon, Canal Zone, ALR.
Reagan, Daniel Joseph, 507 Main St., Worcester 8, Mass., OP.
Reddy, John Bernard, U. S. Naval Hospital, Annapolis, Md., ALR.
Reitz, Russell E., 446 Linwood Ave., Buffalo 9, N. Y., OP.
Reynolds, Garland Alexander, 826 Themis St., Cape Girardeau, Mo., ALR.
Richardson, George S., 201 S. Arno St., Albuquerque, N. M., ALR.
Richardson, James Mark, 6306 Cottage Grove Ave., Chicago 37, Ill., OP.
Richardson, Oscar B., 880 Bay St., Toronto 5, Ont., Canada, OP.
Rockwell, Albert George, Jr., 300 Homer Ave., Palo Alto, Calif., ALR.
Romano, John Emil, 4010 W. Madison, Chicago 24, Ill., ALR.
Rooker, Richard W., 423 Walnut Ave., Niagara Falls, N. Y., ALR.
Ross, Maurice E., 535-E Grand, Beloit, Wis., ALR.
Rothman, Harold, 41 Eastern Parkway, Brooklyn 17, N. Y., OP.
Russell, William Marler, New Bank Asheville Bldg., Asheville, N. C., ALR.
Ryan, Robert Emmett, 3903 Olive St., St. Louis 8, Mo., ALR.

Sacks-Wilner, Erwin Preston, 225 W. State St., Trenton 8, N. J., OP.
Sanderson, Bruce A., 2575 E. Eighth St., National City, Calif., ALR.
Santamarina, Fernando Garcia, Calle 13 No. 154, entre L y K, Vedado, Havana, Cuba, ALR.
Saunders, Joseph Hamilton, 288 S. Limestone St., Lexington, Ky., OP.
Scheer, Alan Austin, 522 West End Ave., New York 24, N. Y., ALR.

Schiff, Maurice, 5459 Diamond St., Philadelphia 3, Pa., ALR.

Schillinger, Robert John, 727 W. Seventh St., Los Angeles 14, Calif., OP.

Schuknecht, Harold F., 950 E. 59th St., Chicago 37, Ill., ALR.

Schutz, William Jack, 672 Francis Bldg., Louisville 2, Ky., OP.

Shafer, Donald McKay, 140 E. 54th St., New York 22, N. Y., OP.

Shaffer, Robert Nesbit, 490 Post St., San Francisco 2, Calif., OP.

Shepherd, Edwin McRae, 1106 Virginia St., Charleston 1, W. Va., OP.

Sherman, Henry Knapp, 121 University Place, Pittsburgh 13, Pa., ALR.

Shier, Julius Milton, 585 Main Ave., Passaic, N. J., O.P.

Shofstall, William Howard, 300 W. 47th St., Kansas City, Mo., ALR.

Simmons, Frederick H., 520 Whites Ave., Marion, Ind., ALR.

Simses, John P., 144 Golden Hill St., Bridgeport 3, Conn., OP.

Skolnik, Emanuel Mitchell, 55 E. Washington St., Chicago 2, Ill., ALR.

Smith, Graham Gable, 304 Doctors' Bldg., Minneapolis 2, Minn., ALR.

Smith, Hal Waugh, Second & "G" Sts., San Rafael, Calif., ALR.

Smith, Joseph George, 490 Post St., San Francisco 2, Calif., OP.

Smith, Trent W., 345 E. State St., Columbus, Ohio, ALR.

Spencer, James Avery, 135 Monte Vista, Watsonville, Calif., OP.

Spencer, James Thomas, Jr., 1112 Virginia St., E., Charleston 1, W. Va., ALR.

Spiro, Barbara, 7449 Cottage Grove Ave., Chicago 19, Ill., OP.

Springer, Kurt C., 504 Arcade Bldg., Kankakee, Ill., ALR.

Stack, David Rodney, Jr., 641 David Whitney Bldg., Detroit 26, Mich., ALR.

Stancil, James Rose, Medical Dental Center Bldg., Bellingham, Wash., ALR.

Stanfill Charles Mac, Medical Arts Bldg., El Paso, Texas, ALR.

Steffensen, Ellis H., Henry Ford Hospital, Detroit 2, Mich., OP.

Steiner, Albert, 1308 Eutaw Place, Baltimore 17, Md., ALR.

Stillerman, Manuel Leon, 109 N. Wabash Ave., Chicago 2, Ill., OP.

Stone, Vean Melford, 3616 Main St., Riverside, Calif., OP.

Stonehill, Alfred A., 6 N. Michigan Ave., Chicago 2, Ill., OP.

Stuart, Edwin Alexander, 1390 Sherbrooke St. W., Montreal, Que., Canada, ALR.

Sun, Kuei Shu, Box 451, Ames, Iowa, OP.

Tabb, Harold Granberry, 1124 Maison Blanche Bldg., New Orleans 16, La., ALR.

Taylor, George Dekle, 111 W. Adams St., Jacksonville, Fla., ALR.

Teitgen, Ralph Emil, Mayo Clinic, Rochester, Minn., OP.

Thompson, Floyd Forrest, 1301 N. Broadway, Santa Ana, Calif., ALR.

Thumim, Mark, 121 Main St., Middletown, Conn., ALR.

Tibbetts, Otis Benson, 33 Court St., Auburn, Maine, OP.

Titche, Leon L., Veterans Administration Hospital, Tucson, Ariz., ALR.

Trent, Robert Irvine, 708 Medical Arts Bldg., Oklahoma City 2, Okla., OP.

Trombetta, Alessandro, U. S. Naval Hospital, San Diego 34, Calif., ALR.

Trotter, John H., 212 High St., Morgantown, W. Va., OP.

Turley, John C., 899 Madison, Memphis, Tenn., ALR.

Ulvestad, Harold S., 202 Doctors' Bldg., Minneapolis 2, Minn., ALR.

Underwood, Ernest Arthur, 601 Main St., Vancouver, Wash., OP.

Virant, John A., 906 Olive St., St. Louis 1, Mo., ALR.

Voorhees, Charles Hammell, 308 College Ave., Elmira, N. Y., OP.

Wainstock, Michael Allen, 1508 David Broderick St., Detroit 26, Mich., OP.

Walker, Donald H., Dollar Title Bldg., Sharon, Pa., ALR.

Walker, James S., 602 W. University Ave., Urbana, Ill., ALR.

Waters, Zack James, 220 Camden Ave., Salisbury, Md., ALR.

Weih, Jack E., c/o Dr. J. G. Beall, 118½ E. Front St., Traverse City, Mich., OP.

Weinstein, Francis Saul, 840 S. 11th St., Newark 8, N. J., ALR.

Weisman, Edward, 2336 Coney Island Drive, Brooklyn 23, N. Y., OP.

Wells, Aubrey Hanson, 400 Hart-Albin Bldg., Billings, Mont., ALR.

West, George Brooks, Jr., 9 Race St., Cambridge, Md., ALR.

West, Stephen Lewis, 109 W. Main St., Taylorville, Ill., ALR.

Westsmith, Richard Alan, 30 S. El Camino Real, San Mateo, Calif., OP.
Wexler, Manuel R., 1917 Wilshire Blvd., Los Angeles 5, Calif., ALR.
Whitaker, Charles Frederic, 55 E. Washington St., Chicago 2, Ill., ALR.
White, Irving Leonard, 812 Pine Ave., Long Beach 13, Calif., ALR.
Wiesenthal, Fred, 30-77 36th St., Astoria, N. Y., OP.
Wiesinger, Warren Edward, 3022 E. 14th St., Oakland, Calif., ALR.
Wolff, Joachim Berthold, 125 E. 84th St., New York 28, N. Y., OP.
Wolkowicz, Michal I., 2959 Richmond St., Philadelphia 34, Pa., OP.
Wright, Joseph William, Jr., 301 Hume-Mansur Bldg., Indianapolis 4, Ind., ALR.

Zurik, Samuel, 3706 Prytania St., New Orleans 12, La., ALR.

DR. WOODS: The Council has received a request from Dr. Frank J. Costenbader, secretary of the American Orthoptic Council, that the Academy publish the papers from the special scientific program of the American Association of Orthoptic Technicians and the American Orthoptic Council, with certain additional material which is to be reprinted and assembled as the *American Orthoptic Journal*. The Council recommends the appointment of Dr. Derrick Vail and Dr. W. L. Benedict as a committee to explore the question, with power to act.

On motion of Dr. Shea, seconded by Dr. Allen, the recommendation of the Council was approved.

DR. WOODS: The Council recommends that the so-called Research Fund be renamed "Educational Fund."

On motion of Dr. Shea, seconded by Dr. Allen, the recommendation of the Council was accepted.

DR. WOODS: The Council was requested to accept the management of a fund to be known as "The Isabella Wilson Research and Educational Fund for Frontal Sinus Pathology," initiated by Dr. Robert H. Fraser of Battle Creek, Michigan. The purpose of this fund is to pay the clerical help in review of case records from the office of Dr. Likely Simpson of Memphis. The Council recommends that this request be accepted.

Upon motion by Dr. McCaskey, seconded by Dr. Vail, the recommendation of the Council was accepted.

DR. WOODS: The Council recommends the election of the following officers for the year 1951:

President: Dr. Derrick Vail
President-Elect: Dr. James Milton Robb
First Vice-President: Dr. Francis E. LeJeune
Second Vice-President: Dr. Peter Kronfeld
Third Vice-President: Dr. Frederick A. Figi
Councillor: Dr. John H. Dunnington
Executive Secretary-Treasurer: Dr. William L. Benedict
Secretary for Ophthalmology: Dr. Algernon B. Reese
Secretary for Otolaryngology: Dr. Howard P. House
Secretary for Instruction in Ophthalmology: Dr. A. D. Ruedemann
Secretary for Instruction in Otolaryngology and Maxillofacial Surgery: Dr. Dean M. Lierle
Secretary for Home Study Courses: Dr. Lawrence R. Boies
Secretary for Public Relations: Dr. Erling W. Hansen

Upon motion by Dr. Mullen, seconded by Dr. Allen, the nominations were closed and the nominations recommended by the Council were accepted.

DR. WOODS: The Council recommends that the next scientific session of the Academy be held at the Palmer House, Chicago, October 14 to 19, 1951.

Upon motion by Dr. McCaskey, seconded by Dr. Vail, the recommendation of the Council was accepted.

DR. WOODS: The Council recommends the following revision of Article V, Section 6 of the By-Laws:

(Following identification of Junior Fellows) Inactive Fellows: Senior or Junior Fellows who by reason of physical infirmity or retirement from practice may, by action of the Council, be classified as "Inactive." Inactive Fellows shall pay no dues, have no voting privileges, and shall not receive the TRANSACTIONS.

On motion of Dr. Vail, seconded by Dr. Allen, the recommendation of the Council was approved.

DR. WOODS: I have nothing further to report.

DR. BROWN: A motion to adopt the entire report is in order.

On motion by Dr. Allen, seconded by Dr. Vail, the report of the senior member of the Council was accepted and the recommendations of the Council were approved.

New Business

DR. BROWN: Is there any new business?

DR. BENEDICT: No new business has been presented to this table.

DR. ALLEN: Dr. Silva and Dr. Sanchez Bulnes from Mexico City have been sent here as representatives of the Fourth Pan-American Congress of Ophthalmology which is to be held in Mexico City, January 7 to 12, 1952, and they wish to extend a cordial invitation to all members to be present.

DR. BROWN: Thank you.

Since no other new business was presented, Dr. Brown introduced to the Academy its new President, Dr. Derrick Vail.

DR. VAIL: Although you may get sick and tired of hearing me say how much I love the Academy and how much I have enjoyed working all these years for the Academy and for my friends in the Academy, I feel that I cannot let this occasion go by without reiterating what I have already said.

I can promise you that I will do my best to serve you faithfully and that I will do my best to make the regime of my office a success and a pleasurable experience for all of you.

Thank you again for the very great honor you have bestowed upon me.

DR. BROWN: I should like to present the President-Elect, my old friend James Milton Robb.

DR. ROBB: Mr. President and members of this great organization: You have bestowed upon me an abiding honor in naming me President-Elect. I do not know whether anybody who receives this honor ever really feels that he deserves it. It is a great honor, for teaching and education are essential factors in the whole problem of medicine. I accept it with all humility.

DR. BROWN: I think we are all grateful to those who have helped make this meeting of the Academy a success, but I personally forgot the Council—men like Dr. Woods, Dr. McCaskey and the others who have done such wonderful work and have helped in every way possible. I want to thank them all for what they have done.

The meeting is now adjourned.

COMMITTEE REPORTS

Upon recommendation by the Activities Committee, the Council voted acceptance of the following reports of standing and joint committees.

REPORT OF THE ADVISORY COMMITTEE TO THE REGISTRIES OF PATHOLOGY

BRITTAIN F. PAYNE, M.D., Chairman

Work on the revision of the Atlases of Ophthalmology and Otolaryngology has reached a satisfactory stage and it is predicted that both will be published before the Academy meets in 1951.

The advisory mission of this Committee will be completed with its meeting October 8, 1950. Final suggestions will be discussed with members of the Armed Forces Institute of Pathology to accelerate publication of the volumes. A report of this meeting will be filed with the secretary of the Academy.

Since the Committee has completed its work the Council is respectfully requested to discharge it with a letter of appreciation from the secretary. It is suggested that the chairman of the Committee, representing ophthalmology, and Dr. Fowler, subchairman for otolaryngology, be retained for administrative purposes until the revision is completed. The advice of former members of the Committee will be asked as the need arises.

A tentative agreement has been made with Saunders and Company, through the Secretary of the Academy, to publish the Ophthalmic Atlas. With the exception of three chapters, the text has been written but not completely edited. The illustrations are being arranged and prepared according to the publisher's specifications.

The illustrations for the Atlas of Otolaryngology are being revised by Dr. Muriel Raum under the direction of Colonel Ash and Dr. Stacy Guild. Her work is most satisfactory but she will need almost another year to complete the work, according to Dr. Fowler. Dr. Raum receives $291.67 each month from the Academy.

Approximately $5,000.00 will be needed to complete the work on the two Atlases.

A brief statement of expenditures for the period from August 31, 1949, to September 1, 1950, follows:

Salaries:

Dr. Muriel Raum	$2,508.32
Mrs. Helen Knight Steward	375.00
Marjorie Davis	372.48
Margaret M. MacFadyen	252.22
Incidentals	143.50
Total	$3,651.52

Reports from Colonel Ash and Dr. Hugh D. Grady, Scientific Director, Armed Forces Registries of Pathology, are submitted for the information of the Council.

Report of Hugh A. Grady, M.D.:

THE AMERICAN REGISTRY OF PATHOLOGY

	Number of cases December 31, 1949
Registry of Ophthalmic Pathology	28,855
Registry of Otolaryngic Pathology	3,375

During calendar year 1949, 292 cases were deleted from the Otolaryngic Registry and 265 cases were transferred to other Registries.

Publications sold during calendar year 1949:	
Otolaryngic Pathology	181
Ophthalmic Pathology	297
Loan sets used during the calendar year 1949:	
Ophthalmic Pathology	128
Otolaryngic Pathology	199

REPORT OF THE COMMITTEE ON THE CONSERVATION OF HEARING

DEAN M. LIERLE, M.D., Chairman

During the past year the Committee on the Conservation of Hearing of the American Academy of Ophthalmology and Otolaryngology held two meetings in Chicago—one during the Academy convention, October 1949, and the other in April 1950.

The activities of the Committee are as follows:

1. Considerable progress has been made in the research project on noise in industry. We are enclosing a copy of the reports of Dr. Grove, chairman of the subcommittee, and Dr. Wheeler, research investigator, concerning the work that has been accomplished.

2. A new motion picture scenario on the problem of hearing has been written, but the production has been delayed because of the lack of equipment.

3. The question of unification of hearing organizations was discussed and Dr. Canfield was appointed to make a survey of the economic need for unification, for which the Audiology Foundation granted the sum of $500. In view of the fact that the Academy's interests are primarily in the field of education and research, no definite action has been taken at this time.

4. The committee on the requirements for training of audiologists formulated by-laws for the establishment of an "American Registry of Audiometrists" and prepared the "Essentials of an Acceptable School for Audiometrists" which were submitted to the Council on Medical Education and Hospitals of the American Medical Association at the meeting in San Francisco in June 1950. A second hearing before the Council will be held in Cleveland in January 1951.

5. A number of new projects have been outlined and work has been started on the following:

a. Manual for the otolaryngologist which will have as its aim, "Raising the Standards of Audiometric Testing and Selection of the Proper Hearing Aid."
b. Syllabus on the Testing of Hearing in Young Children.
c. Manual for Organization of State and Local Conservation of Hearing programs.
d. Revision of the Newhart Syllabus of ministration of a Program for the Audiometric Procedures in the Ad-Conservation of Hearing of School Children.

6. Dr. Henry L. Williams of the Mayo Clinic, Rochester, Minn., has been appointed as a new member of the Committee to succeed Dr. Ernest Seydell, who resigned.

7. We feel that marked progress has been made, and the Committee on the Conservation of Hearing respectfully recommends that the American Academy of Ophthalmology and Otolaryngology continue its financial support to the extent of granting $6,000 for the next year in order that Dr. Wheeler may further pursue the research on noise in industry. In addition, $3,000 will be needed for expenses and other activities of the Committee.

Report of W. E. Grove, M.D.:

THE SUBCOMMITTEE ON NOISE IN INDUSTRY

During the time since the October 1949 meeting of the Committee on the Conservation of Hearing, the work of the subcommittee has been somewhat handicapped by the indisposition of the chairman. Nevertheless, Dr. Douglas Wheeler has been actively working for the most part on the West Coast in conjunction with Dr. House, with Mr. Cantor of the Surgical Mechanical Supply Company, and with the Navy installations at Terminal Island and Mare Island. In conjunction with Mr. Cantor, and also with Dr. Reger, considerable work has been done by him on new types of ear defenders both of the ear plug type of obturator and of the ear muff or helmet type. During the past few weeks Dr. Wheeler has been working in Milwaukee, first with the Chain Belt Company, where a preemployment hearing testing program has actually been set up and is in operation, and also with the health and safety department of the Allis Chalmers Manufacturing Company, where such a program is still in contemplation. While this program at the Allis Chalmers Manufacturing Company is not yet in active operation, it has been ordered by the higher-ups, and Dr. Wheeler has been requested to return there in June. Dr. Wheeler's salary is being paid by the Academy. His working expenses are being paid by a $5,000 grant from the engineering committee of the Mutual Casualty Association. We hope that these insurance carriers will continue this support for the next few years.

A growing interest in the subject of noise and hearing is being advanced by the public as evidenced by articles appearing in the lay press and by inquiries from manufacturers and their agents reaching the office of the chairman and also in the mail of Dr. Wheeler. Our mailing list has expanded from around 1,200 to about 1,700, and in the near future reprints of papers by Dr. Wheeler and Dr. Guild will be mailed out.

In February of this year a round table discussion of the subject of noise and hearing was set up by the Council on Industrial Health of the American Medical Association, and at this conference Drs. Wheeler, Hoople, and Guild took part.

The American Hearing Society has set aside one afternoon at its next meeting in Chicago in June for a symposium on the subject of industrial deafness on which Dr. Wheeler, Dr. Nash and I will appear.

A meeting was held by the Subcommittee on Noise in Industry at the Palmer House, Chicago, Ill., April 30, 1950 at 9:00 a.m. The following were present, Drs. Hoople, House, Guild, Wheeler and Grove. Dr. Guild presented a report of the joint conference held at the Roosevelt Hotel in New York between the Council on Industrial Health of the A.M.A. and the Subcommittee on Noise in Industry at which he acted as a co-chairman together with Dr. James Sterner of Rochester, N. Y., representative of the Council on Industrial Health. This report had originally been written by Dr. Sterner and briefly revised by Dr. Guild. This report is to be published and the reprints sent out to industrial hygienists all over the country. It should be very good propaganda. Dr. House discussed the possibility of investigation of the production of sound pictures illustrating the hazards of excessive noise as far as the hearing is concerned, and it was felt that possibly the Union Pacific Railroad or some other large plant employing labor where noise was a hazard might be interested in financing such a project.

It was also suggested by the members of the subcommittee that a letter be drafted and sent to the otologists of the country outlining the facts concerning the problem of industrial loss of hearing that should appear in specific reports such as otologists address to insurance carriers. It was believed that at the present time otologists cannot give any positive authoritative answers to the question of whether noise has damaged an individual's hearing unless certain factors are known, such as:

1. The pre-employment status of the hearing
2. The noise level of the individual's job
3. The length of daily exposure to that noise level
4. The total length of exposure to that noise level
5. The protection devices supplied and used
6. The possibility of other factors in operation to reduce the hearing

II

Dr. Douglas Wheeler has accomplished a great deal on the research work on the West Coast and he has presented the following report:

At the present time, this Committee has access to organizations representing four divisions of industry: (a) aircraft production, (b) shipyards, (c) railroads, and (d) fabrication of heavy metals.

Aircraft	*Shipyards*
Lockheed, Douglas	Terminal Island
North American	Mare Island
American Helicopter	
Heavy Metals	*Railroads*
Allis Chalmers	Union Pacific
Chain Belt	

The Lockheed Company has a well organized hearing conservation program, in which all new employees are routinely checked by audiometry as part of the initial medical examination and all employees in excessive noise areas are checked every month or oftener, if indicated. These data are available to the Committee. North American is giving us excellent cooperation. In addition to a hearing conservation program similar to that at Lockheed, this company is welcoming research; we have an experimental study in progress now, with several more to follow. Recently, arrangements have been concluded with Douglas Aircraft to begin research and to assist in the development of its conservation program. American Helicopter, a smaller company, has invited inspection for the purpose of determining the seriousness of its noise problem.

The Terminal Island facilities are now largely inactive, although a maintenance staff is still present. The authorities at Mare Island have welcomed the work of this Committee and made all facilities available for study. The plan of research originally designed for Terminal Island has been transferred to Mare Island and an initial survey of the apprentice group conducted.

Dr. House has made arrangements with Union Pacific for a study of noise levels prevailing in diesel locomotives under heavy load. This railroad is also interested in noise and hearing problems around its freight yards.

Recently two companies in Milwaukee have undertaken a hearing conservation program. Chain Belt has added audiometry to the medical examination of new employees. The Allis Chalmers Company has submitted a proposed program, based on recommendations from this Committee. Their safety engineer hopes to visit Iowa for instruction under Dr. Reger; he will select and have trained a suitable audiometrist. Dr. Grove and I expect to visit Allis Chalmers again in June, at which time it is hoped that the program will be under way.

This summarizes the locations from which data either are already available or are to be expected in the near future. The evidence from the mailing list indicates, potentially, a

more diversified interest. This Committee has received correspondence from companies representing the following divisions of industry: steel, farm machinery, chemicals, elevators, automobiles, light metals, arsenals, glass, mining, food packaging, boiler plate and paper refining.

In Los Angeles, two agencies have given evidence of interest in the work of this Committee. Aircraft Industries Association, which is composed of management representatives, engineers, and medical directors from the various aircraft companies on the West Coast, maintains contact with similar companies all over the country. The general purpose of this organization is to share information on common problems, including those involving aeromedical subjects. A division of this Association is the Sub-Committee on Noise and Vibration, a group with which Dr. House and I have met on several occasions. Through this latter agency, we have received important data on noise and vibration in air frames and from motors. Recently, the AIA held a joint conference with the Air Materiel Command, attended by engineers from every major aircraft company in the country. The conference dealt with noise as a nuisance and a public liability. This Committee was given a place on the program; acting for Dr. House, I described in general the work of the Committee on Conservation of Hearing through its subcommittee on noise in industry.

Aircraft Manufacturers Safety Council is composed of industrial relations officers and safety engineers from the West Coast aircraft industry. In January, I presented the views of the Committee on Conservation of Hearing in a speech before this group; I have been asked to return when additional data are released by our Committee.

This Committee now has contact with the Aero Medical Laboratory at Wright-Patterson Air Force Base, Dayton, Ohio. The Laboratory is studying acoustic mufflers and will also take up the matter of ear defenders. Dr. H. O. Parrask, Chief of the Bio-Acoustic Unit, is prepared to exchange information with this Committee.

Correspondence with Mr. L. P. Walters, loss prevention research engineer for Hardware Mutual, indicates that certain companies insured by this carrier may be willing to entertain research on noise problems. Mr. Walters has mentioned several specific cases. Dr. Grove and I expect to confer with him in June.

Through Dr. Grove, I met and talked with Dr. E. G. Meiter, industrial hygiene director for Employers Mutual. Dr. Meiter believes that heavy industry in the Milwaukee area will soon be more receptive to investigation of the noise problem. He would like us to meet with insurance company representatives there and suggests that such a meeting be arranged during June.

Predictive Tests

A. Administration

One experimental form of this test has been administered to apprentices at Terminal Island and Mare Island. These groups were selected for four reasons:

1. A sufficient number of normally hearing subjects could be obtained.
2. These men are studying their trades in a program which offers the possibility of some experimental control.
3. Noise exposure is relatively high.
4. There will be very little turnover among these subjects for at least three years.

The results of the test given indicate that more information is needed about the application of the test principle. The data already collected will continue to be useful, but it appears that their interpretation will depend upon additional data; these data may best be collected under laboratory conditions. A list of the suggested experimental data required is given at the close of this report.

B. Laboratory Studies

There is a good possibility that the necessary studies can be conducted at the State University of Iowa and at the University of Southern California. Dr. Reger and I have discussed the problem at some length; Dr. Charles Lightfoot at the University of Southern California is also interested. In all probability, some aspects of the test development will be improved by application of the subject-controlled audiometer. Dr. Reger has designs for such an audiometer; a somewhat different form may be made available by the Allison Laboratories in Los Angeles.

Ear Defenders

The work on ear defenders may arbitrarily be divided into research and development.

A. Research

A good deal of attention has been given to the study of basic materials and technics for occluding the ear. Some of this work has already been reported before this Committee. Because unusual difficulty has been encountered in creating effective occlusion, the possibility arose that some alternate pathway might be present, by-passing the obstruction in, around,

or over the meatus. One such pathway might be the eustachian tube. I spent several days at the Walter Reed General Hospital in Washington, and while there Dr. Aram Glorig and I attempted to measure the relative efficiency of the eustachian canal in air conduction. The results of our work indicate that the eustachian pathway is probably not important to the problem of occlusion.

The study of basic materials and technics has, as yet, disclosed no essentially new principles. In general, it may be said that the best occlusion measured to date was obtained by a technic difficult to apply practically. It also appears that the effect of mass as a factor in occlusion must be re-evaluated.

B. Development

The objectives in development of an improved ear defender are still, as previously reported, comfort, effectiveness, and low cost. It has been possible to improve ear effectiveness of the standard SMR plug, as will be shown. A new design, however, produces better occlusion and is aimed at meeting some of the objections brought against plugs.

Research Data Required for Predictive Test

Physical—the exposure stimulus and conditions
- a. Simple
- b. Complex
- c. Length of exposure
- d. Level of exposure
- e. Continuous or repeated exposure

Psychological—subject response measures
- a. The critical frequency or frequencies
- b. The rate of onset of threshold shift
- c. Maximum shift in decibels required
- d. Rate of recovery

Problems—to be resolved by application of above data
- a. The optimum choice among the above variables
- b. Reliability of selected measures on normal ears

When these facts have been satisfactorily determined, the test may be standardized on a sample of unexposed (nonindustrial) normal ears. Validation still depends upon application in the industrial situation.

REPORT OF THE COMMITTEE ON SCIENTIFIC EXHIBIT

KENNETH L. ROPER, M.D., Chairman

I was invited to attend the meeting of the Program Committee which met in Chicago on January 14 and 15, 1950, and also the Board of Secretaries meeting on May 28, both of which I attended.

Immediately after the meeting of the Program Committee in January, application forms were sent out to prospective exhibitors. Twenty-four exhibits were accepted for this year's Scientific Exhibit.

A diagram showing the layout of the entire Scientific Exhibit was prepared, and printed copies of the layout were ordered through the Academy office. A copy of this diagram was sent to each exhibitor along with a letter of acceptance under date of June 1. Information regarding these exhibits was forwarded to the Academy office for publication purposes.

Eight of the applications for exhibits received were not considered. Eight additional applications for exhibits, while regarded as worth while, were received too late for consideration and allotment of space and are to be carried over for consideration by the Committee on Scientific Exhibit for the 1951 meeting.

REPORT OF COMMITTEE ON STANDARDIZATION OF TONOMETERS

JONAS S. FRIEDENWALD, M.D., Chairman

Testing Stations

Three testing stations have continued in operation during the past year, directed respectively by Drs. Posner, Kronfeld, and Harrington. The statistical summary of their activities is given in Table I.

Certification of Tonometers

Certification of tonometers by the Electrical Testing Laboratories, Inc., has been continued. Two hundred and sixty instruments were tested and certified during the year July 1, 1949 to June 30, 1950.

During the past year arrangements for the certification of the new model Gradle tonometer have been worked out and a calibration scale for this instrument supplied to the manufacturers.

Special Investigations

Work on the basic calibration of tonometers, on the effect of general anesthesia on tonometry, and on the calibration of tonometers for buphthalmic and microphthalmic eyes is continuing.

Exhibits

An exhibit on the methods used in standardization of tonometers was prepared for the

Pan-American Conference on Ophthalmology in Miami and a more extensive exhibit was presented to the International Congress of Ophthalmology in London in July.

International Standardization of Tonometers

Your Committee has recommended to the Concilium Ophthalmologicum consideration of the formulation of internationally acceptable standards for tonometers and has offered its assistance in working out the technical details.

TABLE I

TONOMETERS SUBMITTED TO TESTING STATIONS

July 1, 1949 - June 30, 1950

TESTING STATION	NUMBER RECEIVED	SATISFACTORY	STANDARDIZED	REPAIRED AND STANDARIZED	REJECTED
Dr. Kronfeld Chicago	188		165	9	14
Dr. Harrington San Francisco	28	2	16	2	8
Dr. Posner New York	98	3	75	12	8

EXPENSES

July 1, 1949 - June 30, 1950

Testing Stations:	
Chicago	$818.61
San Francisco	184.00
New York	260.16
Part Time Secretary and Technician	460.00
Office Expenses	22.54
Miscellaneous	10.00
Total	$1,755.31

REPORT OF THE COMMITTEE ON THE PREVENTION OF BLINDNESS

RALPH I. LLOYD, M.D., Chairman

Our function is to cooperate with the National Society for Prevention of Blindness and do what we can to further efforts along the lines of prevention. The first question coming before us was whether penicillin should be recommended to take the place of silver nitrate instillations at birth. Dr. Post and I felt that the time had not yet come when a changeover from a reliable method should be made. Dr. Woods felt otherwise. Dr. Post and I felt that there should be a lot more evidence available before we could approve of laying aside a method which had reduced blindness due to ophthalmia neonatorum to a neglible quantity.

The National Society for the Prevention of Blindness held a three-day conference in New York City which the committee had endorsed and to which a subscription of $150 was recommended to the Council. I attended the conference and spoke on behalf of the Academy.

I represented the Academy at the meeting of the Lions Club of New York where scholarships for courses in ophthalmology were awarded. This is an annual custom started by Mr. Fryxell of Halpert & Fryxell, opticians. The courses are given by the New York University Post-Graduate Medical School under Dr. Braley.

I also attended the meeting of the New York Society for Service to the Blind, where the Megil medal was awarded Miss Grace Harper of the New York State Department of the Blind.

REPORT OF THE COMMITTEE FOR THE STUDY OF AUDIO-VISUAL INSTRUCTION

DEAN M. LIERLE, M.D., Chairman

This Committee has had no meeting since the 1949 Academy convention. There was no business pending; consequently, there is no report.

REPORT OF THE ADVISORY COMMITTEE TO THE EYE HEALTH COMMITTEE OF THE AMERICAN STUDENT HEALTH ASSOCIATION

LAWRENCE T. POST, M.D.
Academy Representative

There is no report from the Eye Health Committee of the American Student Health Association as there was no meeting of this committee.

REPORT OF THE AMERICAN ASSOCIATION FOR THE ADVANCEMENT OF SCIENCE

M. ELLIOTT RANDOLPH, M.D.
Academy Representative

In my capacity as representative of the Academy to the American Association for the

Advancement of Science, this is to notify you that during the year 1949-1950, I have been in contact with the Administrative Secretary of the Association. The minutes of the meeting of the Council have been carefully reviewed and your representative feels that there are no matters of sufficient significance to be brought before the Academy.

REPORT OF THE AMERICAN BOARD OF OPHTHALMOLOGY

ALGERNON B. REESE, M.D.
Academy Representative

Since the last meeting of the Academy, two examinations were held by the Board: St. Louis, October 18-22, 1949, and Boston, May 22-26, 1950. A third examination will be held before the next meeting of the Academy, and this will be in Chicago, October 2-6, 1950.

At the St. Louis examination, there were 85 candidates and of these 51 passed, 27 conditioned, and 7 failed. At the Boston examination, there were 149 candidates of which 98 passed, 49 were conditioned, and 2 failed.

A written qualifying test was held on January 13 and 14, 1950, at various places throughout the country. Three hundred thirty-nine candidates took the examination, and of these 151 passed, 141 were conditioned, and 44 failed.

The Board is most grateful for the splendid help of many members of our specialty who have served as associate examiners. Their service is, of course, indispensable and the sacrifice they make is appreciated. Also, the cooperation of the staffs of the institutions at which the examinations are conducted is greatly appreciated.

The officers for 1950 are: chairman, Dr. Algernon B. Reese, New York; vice-chairman, Dr. John H. Dunnington, New York; secretary-treasurer, Dr. Edwin B. Dunphy, Boston.

The examinations scheduled for the future are San Francisco, March 12-16, 1951; New York, May 31, 1951, and Chicago, October 1951 at the time of the Academy meeting.

A new directory listing all diplomates was issued January 1, 1950. To those who have purchased this directory, the annual supplements are sent gratis.

At the Boston meeting, Dr. James H. Allen of New Orleans, La., was elected to the Board as representative from the Section on Ophthalmology of the American Medical Association to serve until December 31, 1954.

During the fiscal year ending April 30, 1950, 273 applications have been received.

The total number of certificates issued to date by the Board is 3,035.

REPORT OF THE AMERICAN BOARD OF OTOLARYNGOLOGY

CARL H. MCCASKEY, M.D.
Academy Representative

During the past year the Board has issued a booklet of information stating the required qualifications of candidates for examination.

It was necessary to hold one extra examination in 1950 to care for a part of the large backlog of applicants who have applied for examination.

The following is a report on the examinations held during 1949 and 1950, with the results of each:

1. 84 examined, 19 per cent failures and conditions, Chicago, Illinois, October 4-7, 1949.
2. 76 examined, 12 per cent failures and conditions, New Orleans, Louisiana, January 8-11, 1950.
3. 79 examined, 28 per cent failures and conditions, San Francisco, California, May 16-19, 1950.

The average percentage of failures and conditions was 19⅔.

The next examination will be held in Chicago, October 2-7, 1950.

REPORT OF AMERICAN COMMITTEE ON OPTICS AND VISUAL PHYSIOLOGY

ALFRED COWAN, M.D.
Academy Representative

During the past year, the various subcommittees have been active. The Committee on the Standardization of Instruments is attempting to establish standards for the development of new ophthalmic equipment. It has been the experience of the American Committee on Optics and Visual Physiology that when standards are established by manufacturers without guidance, they are often faulty and difficult to modify. Also, they are often accompanied by misleading advertising. The Committee is especially concerned about the rather careless way in which ophthalmologists purchase and use instruments for the application of beta radiation, with no further idea of their po-

tency than the salesman's word or an unverified statement of the manufacturer.

This subcommittee is encouraging several other types of research on new instruments, e.g., the development of suitable polaroid vectographs for use with standard visual acuity projectors to aid in determination of the status of the fusion mechanism in routine examinations.

A study is being made in the Clinic at the University of Oregon Medical School to determine the suitability of fluorescent.germicidal units for darkroom lighting and the prevention of the spread of communicable diseases in ophthalmologists' offices.

It has been called to the attention of the subcommittee that there is need for development of several types of apparatus, e.g., an improved haploscopic device which will permit the use of bigger and opaque targets with better control of interpupillary distances and a lessened sensation of nearness than is possible with the presently available major amblyoscopes. It is felt by this subcommittee that the American Committee on Optics and Visual Physiology can serve an important purpose by fostering this type of research and encouraging the development in ophthalmology of more laboratories suitably equipped with optical and electrical equipment to conduct research in visual physiology. At present this type of research is being conducted in only a few clinics and medical schools.

Another subcommittee is still working on the contact lens problems. Not only the scientific, but the commercial and the medicolegal aspects are being discussed at length, so that a valuable contribution may be made. The recommendations made by this subcommittee, which have already been presented, seem excellent suggestions for regulating the prescribing and fitting of contact lenses. If their intent is carefully followed, even if controlling legislation is not forthcoming, there should be a minimum of danger to the patient and there is a good possibility of continuing improvement in these lenses through well directed research.

The National Society for the Prevention of Blindness has sponsored a visual screening test of 1239 school children in the first and sixth grades of public schools in Saint Louis, which has been completed. This investigation was made for the purpose of evaluating the various rival methods proposed for screening school children. The data have been collected and are being subjected to statistical study, upon completion of which and approval by the Committee they will be published.

The Committee on Research in Visual Acuity in Myopia has continued the myopia training project and in the last 27 subjects examined, the time element in assessing visual acuity has been introduced, which should help us in the final evaluation of that method of training. All orthoptic training and manipulation of the eyes has been purposely avoided in the hope of assessing the value of that training in which the speed of perception is stressed. In the last group trained, all subjects showed improved visual acuity at the end of the training period, but it is our belief that a sufficient number of controls is not yet available so that final analysis of the data obtained should not yet be made. We have evidence that the myopia is not changed.

In conclusion, the Committee believes that the problem presented commonly requires extensive research and that this often has to be done by outside workers. The Committee recommends that sufficient funds be made available by the parent societies so that these needed studies may be made under the direct supervision of the Committee and more scientific answers may be obtained.

REPORT OF THE AMERICAN ORTHOPTIC COUNCIL

KENNETH C. SWAN, M.D.
Academy Representative

The fifteenth annual meeting of the American Orthoptic Council was held in Chicago on October 8, 1949, with Drs. Swan, Campion and Costenbader representing the American Academy of Ophthalmology and Otolaryngology.

The outstanding activity of importance for 1949 was the conducting of the second annual Council-sponsored didactic course for orthoptic technicians under the leadership of Dr. Walter B. Lancaster, with the assistance of some of the lecturers and instructors who helped with the 1948 course. This was held in Boston with an enrollment of 20 students. It is hoped that if the demand for centralized, intensive didactic instruction in orthoptics continues, such courses may become an annual event. The course for 1950 is coming along apace and will be held in Boston again for nine weeks during July and August.

The American Orthoptic Council would commend the American Association of Orthoptic Technicians for their increased activities during the past year: the technicians arranged a most attractive booth among the exhibits of

the American Academy of Ophthalmology and Otolaryngology. The Association has established annual meetings of each of four regions, Western, Midwestern, Southern and Northeastern. The programs of these regional meetings were participated in by ophthalmologists and technicians, the programs being of great interest and doing a great deal toward improving the information level of orthoptics.

During the past year certain additional activities interested the American Orthoptic Council:

1. The annual symposium for ophthalmologists and technicians was held at the time of the Academy meetings. The speakers were Dr. Edwin F. Tait, Dr. Robert Hill, and Miss Marjorie Enos. Discussers were Dr. Michel Loutfallah, Miss Electra Healy and Mrs. Louisa Wells Kramer.
2. The following new members were elected to the Council:
   Dr. Frank D. Costenbader from the American Academy of Ophthalmology and Otolaryngology
   Dr. Walter H. Fink from the American Ophthalmological Society
   Dr. Beulah Cushman from the American Ophthalmological Society (to fill the unexpired term of Dr. Derrick Vail, who resigned)
   Dr. LeGrand H. Hardy from the Section of Ophthalmology, American Medical Association
   Dr. Alston Callahan from the American College of Surgeons
   The associate member (technician) elected by the Council was Miss Dorothy Bair.
3. Through the combined efforts of the American Association of Orthoptic Technicians, the American Academy of Ophthalmology and Otolaryngology, and the American Orthoptic Council, instruction courses were made available to the technicians and were well attended at the time of the Academy meetings.
4. The Council would again call to the attention of the American Academy of Ophthalmology and Otolaryngology the generous scholarship offered by the Delta Gamma sorority to help train orthoptic technicians and instructors for the blind. These scholarships have been quite helpful to individual students.

The following officers of the American Orthoptic Council were elected in October 1949: president, Dr. Walter B. Lancaster; vice-president, Dr. Richard G. Scobee; secretary-treasurer, Dr. Frank D. Costenbader.

Your committee respectfully recommends:

1. That the American Academy of Ophthalmology and Otolaryngology continue its sponsorship of the American Orthoptic Council, and that it appoint Dr. Kenneth C. Swan for a period of three years.
2. That the American Academy of Ophthalmology and Otolaryngology continue its financial support of the American Orthoptic Council to the extent of $200 for 1950-51. The expenses of the Council have materially increased with the establishment of the annual training course.

REPORT OF THE CONSULTATIVE PANEL ON TUMOR TERMINOLOGY OF THE AMERICAN SOCIETY OF CLINICAL PATHOLOGISTS

JOHN S. MCGAVIC, M.D.
Academy Representative

I regret to report that the Consultative Panel on Tumor Terminology of the American Society of Clinical Pathologists has taken no action during the past year.

REPORT OF THE HELMHOLTZ CENTENARY COMMITTEE

F. BRUCE FRALICK, M.D., Chairman

The Academy's Helmholtz Centenary Committee has had no formal meeting during the past year. We have had no correspondence from Dr. Burton Chance referable to any further assistance which we might be to him. Since Dr. Chance and his committee from the American Ophthalmological Society initiated this program, it was felt that they should be the leaders and that we should be ready to serve in an advisory capacity or to help them in any way they might suggest.

I have again contacted all of our Committee members and they have no further suggestions to offer other than those which they have transmitted to Dr. Chance in the past year.

REPORT ON THE INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

R. TOWNLEY PATON, M.D.
Academy Representative

The International Organization against Trachoma was founded officially by the Thir-

teenth International Congress of Ophthalmology in 1929. It provides meetings at which all matters connected with trachoma may be discussed.

It was my privilege to attend the last meeting as official delegate from the United States of America. This meeting was held in London on Wednesday, July 19, as part of the program of the Sixteenth International Congress of Ophthalmology. The opening paper on "The Initial Signs of Trachoma" was given by the President of the Society, Dr. A. F. MacCallan, and he was introduced by Dr. Sedan. A general discussion followed and then a number of papers were given on the newer methods of treating trachoma. Perhaps the most interesting of these papers was given by Dr. Tabone of Malta, who reviewed Dr. Alson E. Braley's experiences in the use of aureomycin in treating trachoma and then gave his results. His results confirmed those of other workers in this field, although he remarked that trachoma in the Mediterranean did not respond so dramatically as had been reported by many previous workers. He summed up his experiences by saying that aureomycin is perhaps the most effective drug in trachoma so far if given in adequate doses. He used aureomycin locally and systemically and feels that probably a combination of both methods shows the best response.

Dr. Tabone was inclined to treat trachomatous patients for periods of five days, and cautioned that many patients might require three or four five-day courses of treatment before showing optimum results. He also mentioned that results should not be looked for in less than two weeks, although in many cases improvement subjectively and objectively was dramatic early in the course of treatment. Locally he has employed aureomycin borate instillations as well as aureomycin ointment; more surprising still, he has used 0.5 per cent solution aureomycin hydrochloride without significant signs of irritation. In administering aureomycin by mouth, he felt that a dose of 500 mg. three times a day should be employed.

The papers and discussions confirmed Dr. Maxwell Lyons' observation. Dr. Lyons has used aureomycin locally and systemically as well as a combination of the two methods. He left little doubt in the minds of his listeners that aureomycin is effective in the treatment of trachoma. While he is not ready to give a final opinion, he feels that aureomycin by mouth may be just as effective as the combined administration of the antibiotic. The dosage he employed locally was the instillation of 0.5 per cent solution hourly during the morning hours and again before the patient went to sleep; when administering aureomycin systemically, he prescribed 50 mg. per kilogram per day, divided into two doses. Dr. Lyons continued treatment for a period of about ten days.

It was my good fortune after leaving London to go to Iran and set up an extensive trachoma control program for the country. One city we visited, Dizfoul, had a population of 60,000 persons. The local eye doctor said that 90 per cent of the population had had trachoma or were in the active stages of the disease. During a two-hour ramble about the city many persons, young and old, were stopped on the street, and everyone had evidence of trachoma as far as one could tell in a cursory examination.

A method extensively used in Iran in the treatment of trachoma is electrocoagulation. The native doctors who use this method claim that not only are the follicles completely destroyed with a minimum of scar tissue formation, but that the current penetrates the lids and kills the virus. This method of treating trachoma did not meet with the general approval of ophthalmologists at the London Conference. Electrocoagulation for the treatment of trachoma is an old method, but it is being kept alive in Iran, as the technic is taught to all medical students. After personal observation on several hundred cases, I could only see that coagulation did destroy the follicles and was an easy method for mass treatment. Each and every case after coagulation also received a liberal swabbing of copper sulfate solution.

Anyone interested in keeping abreast of the latest developments in the field of trachoma should subscribe to La Revue International du Trachome. Articles by members may be sent to the editor, Dr. Sedan, 94 Rue Sylvabelle, Marseille. Membership in the Organization is by annual subscription of thirty shillings sterling.

REPORT OF JOINT COMMITTEE ON INDUSTRIAL OPHTHALMOLOGY

A. D. RUEDEMANN, Chairman

The three main objectives of the work of the Joint Committee continue to follow along the lines of research, education and service to industry.

TRANSACTIONS *of the American Academy of Ophthalmology and Otolaryngology*

Important papers on industrial eye problems of interest to members of the profession continue to be a feature of the Section on Industrial Ophthalmology of each issue. Much interest has been stimulated through this medium and reprints of the articles published have been requested by medical directors, nurses, safety and personnel directors of industry.

Bibliography

The bibliography on industrial eye subjects to date contain approximately 3000 references and is enlarged continually in the office of the secretary. Reference material is available at all times upon request and is published currently in the TRANSACTIONS.

Kodachrome Slides (2 x 2)

A library of lantern slides for lecture purposes is being accumulated. A limited number of duplicate slides is already available for loaning purposes.

Consultation Services

Considerable correspondence is carried on by the secretary in response to requests for information in the handling of various problems which have to do with injuries, pathology, medicolegal, testing programs, illumination and safety eye wear. The requests come from home and abroad and any unusual problems are referred to recognized authorities in their respective fields.

Dr. Andrew C. Ivy, vice-president of the University of Illinois School of Medicine; Col. Victor A. Byrnes, and Dr. Hedwig S. Kuhn, secretary of the Committee, will speak at a special scientific program during the Academy meeting. The general subject to be discussed will be "Eyes and Disaster."

Research

1. Development of a thesis on emergency eye care in disaster.

2. A survey of second injury funds in the 48 states in which the National Society for the Prevention of Blindness and the Pan-American Association of Ophthalmology are especially interested.

3. An attempt is being made to evaluate the opportunities for industrial rehabilitation of the blind and to work out a practical plan whereby the ophthalmologist may be better equipped to assist in this important work.

The secretary and another member of the Joint Committee are serving on the Armed Services N. R. C. Vision Committee, a contact which has enriched our awareness of industrial problems considerably.

A panel program was arranged by the secretary for the American Association of Industrial Physicians and Surgeons during their annual convention in April, 1950.

During the past twelve months, the Joint Committee worked directly with the optical companies in an effort to ensure ethical and efficient methods of distributing their products.

Much of the work of publicizing the Manual on Toxic Eye Hazards was handled by the Committee through the secretary's office and the response to the 2500 direct contacts made with ophthalmologists, medical directors and public health officials was very gratifying.

The work of the Committee has been augmented by the generous increase in the grants from the Council of Industrial Health of the American Medical Association and the American Academy of Ophthalmology and Otolaryngology. This will make possible a wider field of opportunity for the projects that are now under way and those being formulated.

Financial Statement:

Joint Committee on Industrial Ophthalmology
September 20, 1950

Balance, Aug. 16, 1949		$ 25.00
Receipts:		
Council on Industrial Health, American Medical Association	500.00	
American Academy of Ophthalmology and Otolaryngology	500.00	
Sale of Manuals on Toxic Eye Hazards	7.00	
		$1,032.00
Disbursements:		
Secretarial, Convention, October 1949	45.36	
Toxic Eye Hazards Manuals on hand	21.00	
Clerical help, mimeographing and postage, Blind Project	51.00	
Postage	203.64	
Plastic mountings for magnifying lens	66.45	
Reprints	47.25	
Subscription to industrial publications for English contacts	50.00	
Negatives made from charts for publication (Dr. Peterson's article)	15.00	
Lantern slides	14.99	
Bank service charges	1.76	
Sallye Rosenberg (Gift for Dr. Ivy)	78.80	
Expense of Dr. Rieke to American Association of Industrial Physicians and Surgeons, April 1950	248.63	
		$843.88
Balance, Sept. 20, 1950		188.12

DIRECTORY OF EYE, EAR, NOSE AND THROAT SOCIETIES

Secretaries of all eye and/or ear, nose, and throat societies — international, national, regional, state, and local — are requested to aid the Academy in keeping up-to-date the Directory of Ophthalmologic and Otolaryngolic Societies, a regular feature of the TRANSACTIONS.

Please supply us with the following information:

Name of society (write out in full).

Names of current officers (president and secretary or corresponding offices).

When elections become effective.

Place of meeting (exact if known, or supply to this office when available).

Date of meeting (exact if known, or monthly, bimonthly, October through May, etc.).

Send to: **W. L. Benedict, M.D., 100 First Ave. Bldg., Rochester, Minn.**

MEDICAL CIVIL DEFENSE: THE BACKGROUND

ANDREW C. IVY, M.D.
CHICAGO, ILL.

MANY of you know that the American Medical Association and the state and city medical societies each have had a committee on civil defense for four or five years. The medical profession realized at the close of the war, and it still realizes, that this country is vulnerable to atomic attack—to a bacterial warfare and chemical warfare—and it has only been in recent years that other people have developed the same opinion.

After the close of the war, I belonged to a group in the city of Chicago which talked about the potential hazards of the atomic bomb used by direct attack or used through the medium of sabotage. We should not forget that it is easy to assemble parts of an atomic bomb, which may then be set to go off at a certain time without being detected. All the makings can be brought in a box and the only way one can find out the contents is to open the box. We not only have to think of that as a possibility but also have to realize that a plane-guided missile or sabotage can result in disaster. The United States Armed Forces warn us that we can expect an attack by any means at any time.

A little over a year ago the mayor of Chicago became concerned over a possible attack on Chicago, for he felt Chicago or any near city would be a strategic target. He then sent a directive to several groups, and one to the Commissioner of Health, Dr. Bundesen, to organize against and prepare for any sort of disaster that might strike Chicago, particularly one that might result from warfare. Dr. Bundeson called together representatives of the medical profession, scientific, chemical and other groups, who had had experience during World War II with atomic and chemical warfare. This constituted the General Committee, and I happened to be selected chairman. In my response to Dr. Bundesen I wrote that I was in favor of the Committee and that I felt it was worthy and in line with my basic general philosophy—to work and hope for the best but to be prepared for the worst. That is the spirit and motive of the people working in the Chicago Emergency Medical Service, one of five divisions.

Of the entire civil defense organization with all its divisions, the Public Health Division has the greatest responsibility in that it has to deal with the human beings living in the attack area. This Committee took as its objective, after studying the problem thoroughly, a plan to decrease the deaths among the nonfatal casualties by 75 per cent. Something *can* be done about an atomic attack in the densely populated city of Chicago. I can prove to you that, by working together and educating and training the public to cooperate with the medical profession, we can achieve that goal. Those two basic conditions are absolutely necessary.

We first set up a committee to develop technics to cover all the problems that might arise from an attack on the city of Chicago. In a catastrophe of this sort, one cannot, outside of hospital practice, permit any variation in the type of treatment given. In the first place, there are not enough physicians

Vice President, University of Illinois School of Medicine.

Presented at the meeting of the Joint Committee on Industrial Ophthalmology, Oct. 10, 1950, Chicago, Ill.

to go around—so we have to get optometrists, chiropodists, biochemical students, dental students, pharmacy students, and so on, who have some knowledge of human anatomy. We have to prepare specific instructions to be given in the training of this personnel. We also will have to train close to 5000 adult lay persons who will become roving first aid teams and who for the first time will be administering drugs such as morphine. Many people don't like to do it; we have to do a number of things we don't like to do. This instruction will be given under Red Cross to first aid classes by the doctors assigned by various branches of the Chicago Medical Society. Specific instructions will be outlined as to the sort of treatment to be given by the roving first aid team, collecting stations, and hospitals. In the third place, we have to be very specific and definite about the treatment that will be required in order that the necessary supplies will be available. One cannot get together all varieties of supplies to take care of various ailments as is generally considered good medical practice. Instead, one must get together basic materials to be put in medical warehouses. Many of these have to be found in the community in which you live. We have a committee surveying the city of Chicago to find out where the medical supplies are, as we may have an atomic attack before we can stockpile gauze, bandages, etc.

The Council appointed committees consisting of leading medical persons in the city of Chicago; these committees have drawn up rules for treatment and have outlined exactly what is to be done at each of the several locations. In the same way, we have asked a small group of ophthalmologists who have had industrial experience to serve as our advisers, and we are relying on this group to classify eye injuries, to decide what is to be done by assistants, and to determine what cases should be sent to the operating sections. Again one has to be very specific in the instructions issued. When the report of the Joint Committee on Industrial Ophthalmology comes in, our Emergency Committee will meet to consider it and to adopt it as policy. It will then be taken before the Chicago Medical Society to be adopted there. In this way we can avoid the confusion that would come as a result of one doctor stating that burns should be treated in a certain way, while another may in a newspaper item recommend another method. All this would be confusing to the public—something we have to avoid.

Medical units consisting of a roving first aid team will be organized under the leadership of a physician and surgeon. For those positions we shall select doctors who have had experience in front line combat and who will not be subject to the draft. Records of all casualties will be kept; in the city of Chicago the accountants are taking over the responsibility of keeping records in first aid and collecting stations. Finally, there must be managers. For those positions we are selecting presidents and executive vice-presidents living at the edge of town who, because of their initiative and resourcefulness, can best organize the first aid stations to be located at the street intersections at the two mile bomb perimeter—i.e., two miles away from the zone of explosion. We will have 340 first aid stations with 88 members on each team, including a carpenter. When we run out of splint material, a carpenter will take over to make splints and, as the need arises, forage lumber from sides of houses or garages.

We will need 600 casualty stations with an average bed capacity of 300 each to take care of the approximately 180,000 casualties. For this we will use public schools, hotels, bowling alleys,

etc. The professional personnel of the Chicago Society of Anesthetists will have to train 6000 people to give and draw blood. The drug stores will be manned twenty-four hours a day by one or two pharmacists at each store. The rest of the pharmacists will be part of the general pool.

The hospitals are now setting up an emergency organization. They will be staffed by a surgeon, assistant surgeon, anesthetist and an aide—all to be on an 18- to 24-hour schedule. Two sets of instruments are to be provided so that one may be in use when the other is being sterilized. We have organized the hospitals on the basis of seniority—the doctors and nurses not needed on the basic teams will become a part of the general pool.

There will be refugee camps where simple fracture cases can be sent, as well as those suffering from simple burns and all those who have been exposed to radiation after they have been decontaminated. Blood counts will be taken in every case in which there has been exposure to radiation.

The last units are the outlying hospitals—within a radius of forty miles of Chicago. These hospitals will also undergo emergency organization, making it their responsibilty to see that the houses in the hospital area are evacuated to provide more space.

For casualties requiring skilled surgery, we will need 28,000 hospital beds in Chicago, and we have asked that double the capacity be provided, using beds in nurses homes and nearby houses.

If there is an attack in the Loop, the first aid collecting station teams will move in, with or without instructions by radio. The chief at each first aid station will have the job of sorting—if the individual is contaminated by radiation and injured he goes to one area, if contaminated and uninjured to another, etc. In that way we will be able to handle within six hours the 180,000 people who will come out of the bombed area. This is the World War II principle of getting the wounded in early and sorting them.

Forty per cent of our doctors will be drafted. The registered nurses in Chicago will have to train 32,000 nurses' aides in an eighty-hour course. In this program the ophthalmologist must take care of all surgical cases. The minor eye injuries will have to be cared for by lay persons who have been instructed in regard to treatment and taught to recognize cases that should be referred for more professional care.

DISASTER STRIKES — WHAT HAPPENS?

COL. VICTOR A. BYRNES (MC)
RANDOLPH FIELD, TEXAS

THE importance of vision in our highly complex civilization can hardly be overemphasized. The loss of vision is a more serious handicap to the individual than is the loss of many other abilities.

The eye is a very critical organ insofar as prompt, proper care following injury is concerned. An injury, which if treated early will give complete restoration of sight, may easily result in loss of the eyeball if treatment is delayed. Improper early care of eye injuries by well meaning friends may also cause the loss of an eye for a patient in spite of the best later efforts of a skilled ophthalmologist.

While the eye makes up only a small portion of the body, its relative injury rate will be high in proportion to its to-

Presented at the meeting of the Joint Committee on Industrial Ophthalmology, Oct. 10, 1950, Chicago, Ill.

tal exposed area. This is true, of course, because a wound or foreign body in the eye can be a serious injury, while the same wound or foreign body would be an insignificant injury almost anywhere else on the body.

It is for these reasons that a program for care of injured eyes in the event of disaster should be formulated. No program can be initiated, however, until the situation expected to exist following a disaster is evaluated. One must know (1) what type of eye injuries will occur, (2) how many eye injuries there will be, (3) who will be available to treat the eye injuries, and (4) what facilities will be available for use (fig. 1).

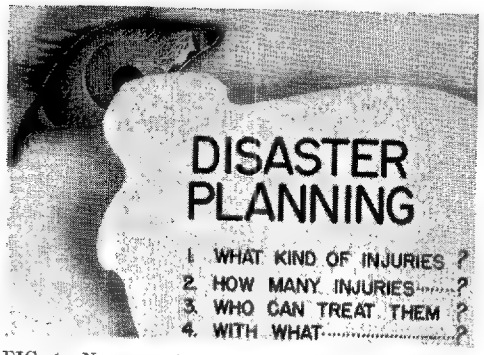

FIG. 1—Necessary information for planning care of any type of casualties.

The types of eye injuries will depend somewhat upon the type of disaster which occurs. Such disasters can be explosions within factories, or they can be larger industrial explosions of the Texas City, Texas, or Amboy, New Jersey, type. Finally, they can be due to bombardment by an unfriendly nation using either high explosives or the atomic type of bombs.

To be prepared adequately for an emergency one must be prepared for the most severe disaster which is at all likely to occur. One is then prepared for any eventuality and anything less than the most severe type can be properly handled. Since the nuclear fission type of bomb represents the most severe disaster that could occur, any program for eye care should be geared to a disaster of this magnitude.

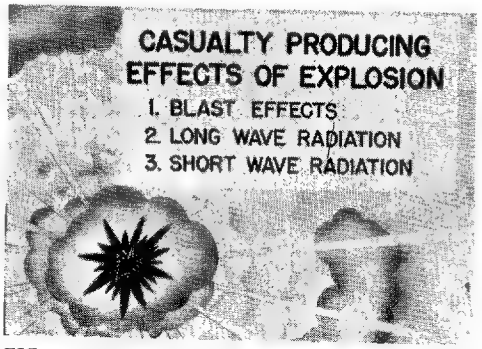

FIG. 2—Casualties produced by any type of explosion are due to one or more of the above factors.

If one considers the factors in explosions which produce injuries (fig. 2), they can be considered under the following headings:

1. *Effects of Blast* (present in all types of explosions)
   a. Direct blast effects on the eye
   b. Secondary effects produced by falling buildings and scattered debris
   c. Displacement effects by which individuals are thrown violently against other objects
2. *Effects of Long Wave Radiation* (heat, including visible and ultraviolet light—present in some degree in all types of explosions)
   a. Flash burns, the direct effect of the radiant energy produced by the explosion
   b. Secondary effects produced by fires started by the explosion
3. *Effects of Short Wave Radiation* (gamma rays, alpha rays, beta particles and neutrons—present only in the atomic type of explosion)

Injuries Due to Short Wave Radiation

Let us consider the last named heading first—injuries due to short-wave radiation. This is the principal factor in an atomic explosion which differentiates it from other explosions. Cogan, Martin and Kimura[2] stated that at Nagasaki and Hiroshima it produced the following effects in the eyes of some of the exposed population:

1. Keratoconjunctivitis with mucopurulent discharge one to three days after exposure. (Probably due to ultraviolet radiation.)
2. Keratitis coming on three to four weeks after exposure.
3. Retinal complications of radiation sickness occurring 10 to 14 days after exposure.
4. Radiation cataracts coming on in a matter of years after exposure.

It is quite obvious that none of these lesions require any emergency eye care at the time of a disaster. For purposes of this discussion we can, therefore, ignore this group of symptoms. This leaves, then, the same characteristics for all explosions except for a difference in degree. Explosions produce acute eye injuries through two mechanisms—heat and blast.

One other point with regard to radiation should be mentioned. It has to do with residual radiation in the bombed area and its effects on rescue personnel. If an atomic bomb were exploded in the air (its position of maximum effectiveness), the residual radiation after the first minute or two would be well within tolerance limits. It would be perfectly safe for rescue parties to enter these bombed areas insofar as radiation is concerned.

Heat and Blast Effects

With radiation eliminated from consideration in the immediate postexplosion period, the discussion can be narrowed to the heat and blast effects. It would be very desirable to know the total number of casualties which would be produced by an atomic explosion, the percentage of this total which would be eye casualties, and the percentage of these eye casualties which would require definitive care. We would further like to know how many would be burned as compared with how many would have traumatic injuries.

Unfortunately, figures on these points are not readily available. In almost all disasters there is such disruption of activities that adequate medical records are not kept. This was true of most of the bombing raids on the continent of Europe in World War II. It is also regrettable for this study that accurate figures on the Hiroshima and Nagasaki atomic bomb explosions are not available. This is true because the chaos that existed following the bombing prevented any accurate study. Even the total numbers of people injured or killed have only been estimated. These estimated totals may vary as much as 30,000. Then, too, early studies which were made had as their primary interest the effects of short-wave radiation on the eyes. They were not designed to study the thermal and traumatic effects. Flick[3] was probably the first American to study these casualties and he states:

> There were large numbers of patients exhibiting facial burns of the ordinary type with dense cicatrix. These burns in some instances involved the conjunctiva and cornea to such an extent that the red, inflamed, lacrimating eyeball had only slight movement and seemed set in a dense mass of contracting scar.

He also stated concerning mechanical injuries:

> Among those noted were penetrating injuries of the globe, leucoma of the cornea, traumatic cataract, various lid deformities, detachment of the retina, and various syndromes involving fractures of the skull and walls of the bony orbit. Many ambulatory casualties were seen in the streets wearing eye patches.

Drs. David Cogan, S. Forrest Martin and Samuel J. Kimura[2] conducted an ophthalmologic survey of atomic bomb survivors in Japan in 1949. They assumed their task was "(1) . . . to determine qualitatively, by history and examination, whether or not ocular lesions, *other than traumatic*, resulted from the atomic bombing. . . ."

The Japanese investigators, Tamura, Ikui, Nakano, Hiwatashi and Oshio,[6,7] Hirose,[4] and Shoji,[5] reported similar findings. In no instance did they attempt to estimate the total number of eye casualties or the percentage of serious eye injuries in relationship to total eye injuries. They, too, were mostly interested in the radiation effects on the eye and the fundus changes which occurred as a result of radiation sickness in the individual. Tamura and his associates made the point that the injuries due to blast effects are essentially the same as other bombardment injuries. The burns of the eyelids were in general not as severe as burns on the faces of the individuals. Perhaps the height of the explosion, plus the oriental type of eyelids, was responsible for this fact. Burns of the cornea and conjunctiva were present but not as frequently as might be expected. They tended to heal with only slight scars. These authors made one very significant notation. They stated that of foreign bodies in the eyes, the most frequent one was window glass. When one considers the relatively greater amount of window glass present in homes in American cities, this particular factor becomes very important.

Because of the lack of available statistical data and because the same factors are operative (though in different intensities), an estimate of probable casualties was attempted by a study of disasters for which more accurate figures are obtainable.

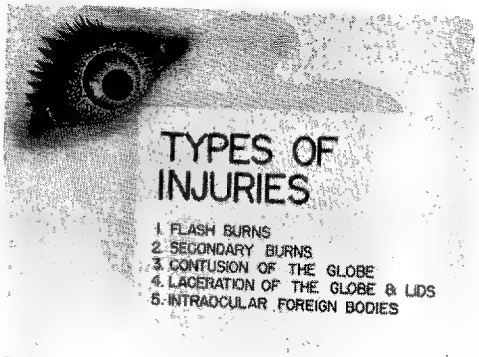

FIG. 3 -The types of injuries of the eye which comprise practically all the eye casualties produced by explosions.

In the event of disaster from any type of explosion the following types of injuries require consideration (fig. 3):

1. Flash burns of the eyelids and eyeball (high intensity infra-red, visible light, and ultraviolet radiation)
2. Secondary burns due to fires started by the explosion
3. Contusions of the globe
4. Lacerations of the globe and lids
5. Intraocular and intraorbital foreign bodies

In order to get statistics on the incidence of injuries in smaller comparable catastrophes, the Texas City disaster of April 1947 was reviewed. Blocker and Blocker[1] have made a tabulation of casualties, which is shown in figure 4.

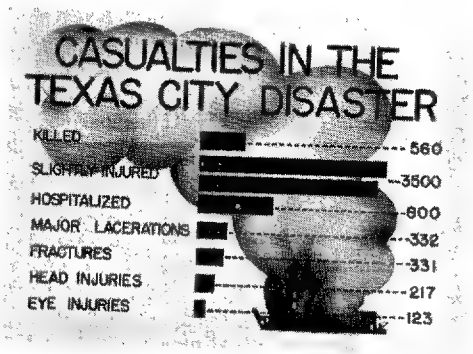

FIG. 4—The number and types of injuries in the Texas City disaster. (From Blocker and Blocker.[1])

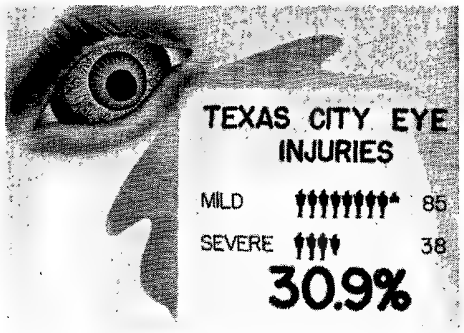

FIG. 5.—The number of severe eye injuries in comparison with total eye injuries in the Texas City disaster.

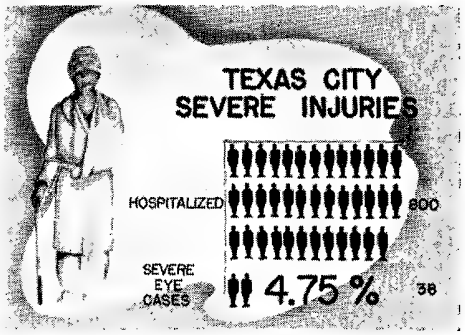

FIG. 7.—The number of severe eye injuries in comparison with the total number of severely injured persons.

In this series, 30.9 per cent of the eye injuries were severe (fig. 5).' If only the 38 severe eye lesions were included in the 800 hospitalized, they make up 4.75 per cent of all hospitalizations. It is probable that more than this were included, thus raising the percentage of hospital beds required for eye patients. It will be noted that eye injuries rank fourth in the number of severe lesions requiring hospitalization.

The total of the slightly injured is assumed to be half way between the 3000 to 4000 figure quoted above, or a total of 3500. Add to this the 800 hospitalized patients, and we have a total injured group of 4300 people. The total number of eye injuries was 123. Thus 2.8 per cent of all individuals injured had eye injuries (fig. 6). In considering only the severe eye injuries we find that 0.87 per cent of all individuals injured were in this category. If the 800 hospitalized cases can be considered to be the serious nonfatal cases, then severe eye lesions make up 4.75 per cent of such seriously injured individuals (fig. 7).

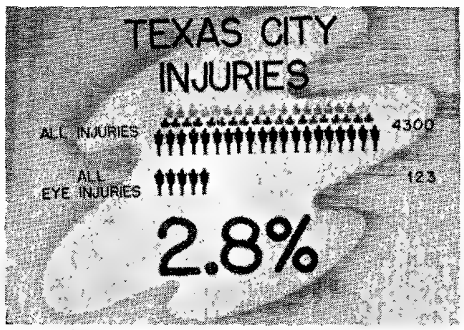

FIG. 6.—The total number of eye injuries in comparison with total of all injuries.

Dr. Gaynelle Robertson of Texas City was kind enough to review the records of all eye patients she could find who were injured in the Texas City disaster. Her review showed the totals for these eye injuries were:

Mild or inadequately recorded	78
Lacerations	3
Burns	8
Loss of eye (1 bilateral)	11
Concussion of eye (1 bilateral)	10
Severe lid or orbital injuries (2 bilateral)	16
Penetrating wounds of eyeball with glass or other foreign body (5 bilateral)	26
Foreign body in orbit	4
Damage to optic nerve	2
Loss of sight, one eye—no details	2
Severe bilateral injuries	10
Mild bilateral injuries	5
Total with at least 1 severe eye injury	44

Her analysis showed that 44 out of 138 cases had at least one severe eye condition, a percentage of 31.8 per cent of all eye cases.

The British made a very careful survey of their casualties occurring in air raids. Their results showed that 7 per

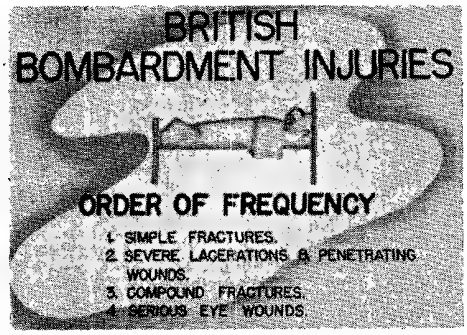

FIG. 8.—The order of frequency of severe injuries incurred in the aerial bombardment of London.

cent of the serious nonfatal casualties had severe eye injuries. Nearly one-third of these had other serious injuries. *Eye injuries were the fourth most common cause of serious injury* (fig. 8).

They found that 2.7 per cent of all air raid casualties had eye injuries. Types of eye injury found in this British study were as follows:

	Percentage
Foreign bodies in the conjunctival sac	31
Lacerations of cornea, sclera and iris	17
Gross damage	14
Abrasions of cornea	12
Subconjunctival hemorrhages	12
Conjunctivitis	6
Hemorrhage in vitreous chamber	5
Blast effects (not serious)	3

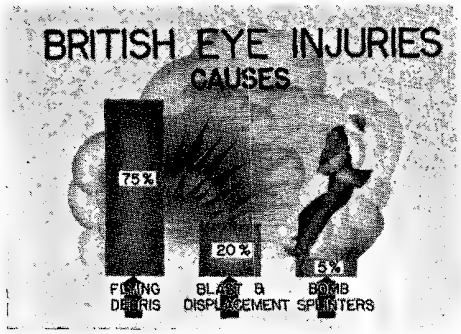

FIG. 9—The direct causes of the eye injuries incurred by the population of London under aerial bombardment.

The causes of these eye injuries are shown in figure 9.

In the later bombardment experience with parachute mines and flying bombs, the incidence of eye injuries was higher, being 11.7 per cent of hospital casualties. They took for a planning figure a total of 10 per cent of hospitalized casualties to be eye cases.

It is interesting to note the similarity in the incidence of eye injuries in the Texas City disaster and in the British reports (fig. 10).

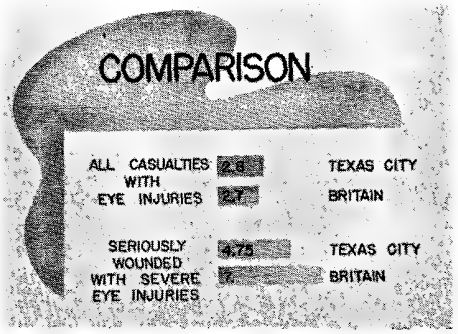

FIG. 10.—A comparison between the British bombardment figures and the Texas City disaster figures.

It is also interesting to note the higher incidence of eye injuries reported in the later study on the more powerful bombs — the parachute mines and the flying bombs. This same trend would be expected with the even more powerful atomic bombs.

These figures cannot be directly applied to the prediction of the incidence of atomic bomb casualties, but there are two things about them which are important. They are essentially injuries due to blast effect since the thermal effect of high explosives is not great. Secondly, they are the type of casualties produced in individuals living in essentially the same types of buildings as those in which we live. For estimation of casualties which might occur here, these figures should be useful.

Estimation of Eye Casualties In an Atomic Raid

The British have informed their people in the newspapers that if an atom bomb struck one of their large cities they would expect not less than 50,000 casualties.

While the Japanese experience is not entirely comparable, we can get a great deal of information from a study of the atomic bomb blast of Hiroshima. Most of the Japanese buildings are flimsier than ours. On the other hand, their better earthquake-proof buildings are stronger than most of our buildings. Their cities have a great deal less glass than American cities. The numbers given in figure 11 indicate the casualties they received from one of the early weaker atomic bombs.[4]

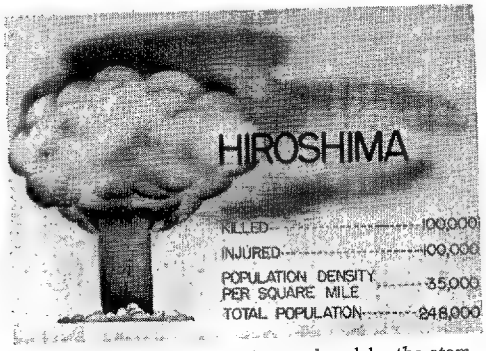

FIG. 11—Estimated casualties produced by the atomic bombing of Hiroshima.

Characteristics of the Atomic Bomb Which Must Be Considered In Casualty Estimation

The short wave radiation effects of an atomic blast have already been discussed and will not be further considered here. The relative amount of heat produced is greater than with other bombs. A higher percentage of casualties will be due to the heat than is true with high explosive bombs. In the bomb burst at Hiroshima it is estimated that 21 billion calories of heat were released

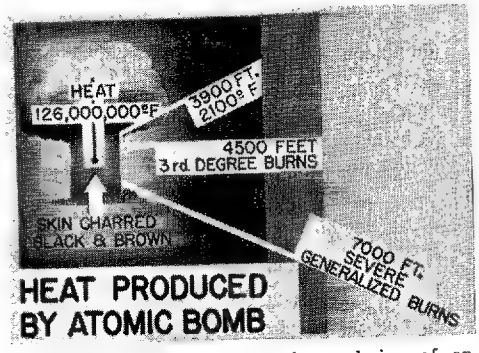

FIG. 12—Heat produced by the explosion of an atomic bomb. It has been estimated that the bomb burst at Hiroshima released 21 billion calories.

and that the temperature at the core of the explosion was 126,000,000 F. (fig. 12). Even as far away as three-fourths of a mile the temperature for a fraction of a second was 2,100 F., which is hot enough to blister tile on roofs. This heat is due to the production of infra-red radiation, which travels at the speed of light. At Hiroshima it produced severe generalized burns at a distance of 7,000 feet. This amount of heat existed for only a fraction of a second, and clothing and the shelter of a building or wall were sufficient protection from it. These direct radiation burns, however, were very severe in unprotected skin. Directly under the blast, skin was charred brown and black by the heat. Serious third degree burns occurred out to 4,500 feet. The available literature does not show the percentage of eye and eyelid burns which one would expect, though Flick[3] mentions severe burns as stated above. A factor of importance is the protection afforded by the orbital ridges and the film of moisture on the cornea. It is known that about 40 per cent of the casualties had flash burns produced by infra-red radiation, or had ordinary thermal burns produced by fires. The exact incidence of eye burns, however, cannot be estimated because there are no figures available for Hiroshima and no comparable figures in other explosions.

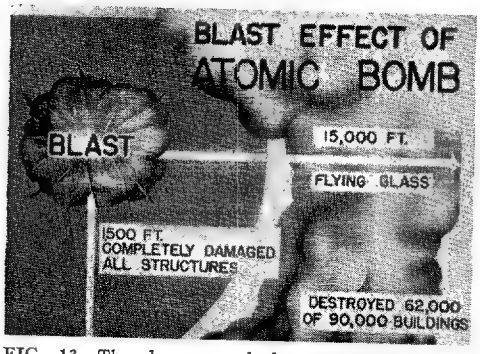

FIG. 13—The damage and destruction caused by the explosion of the bomb at Hiroshima, which occurred at an altitude of 1,500 feet.

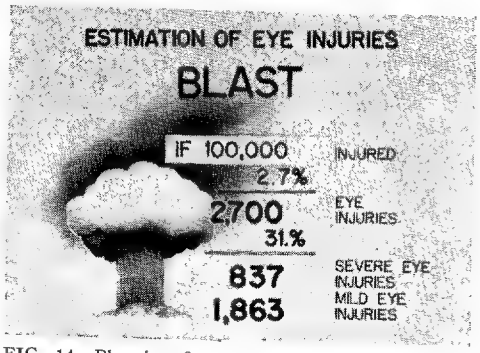

FIG. 14—Planning figures for estimating the number of eye injuries which might be produced in an atomic bomb explosion in an American city.

Blast Effect

The blast lasted about one second. It was not sharp, but was very strong. It didn't punch holes in buildings. It was much more apt to push them over. Of the 90,000 buildings in Hiroshima, 62,000 were destroyed (fig. 13). The blast produced flying glass up to 15,000 feet. Although it produced a relatively small group of casualties by direct effect, it produced many by displacement of people, flying missiles, falling debris, etc. About 40 per cent of the injuries were produced by these forces, although some authorities estimate as high as 70 per cent.

The number of casualties which would be produced in an American city would depend upon the time of day the attack occurred. This would in turn determine the population density in the area attacked and whether or not individuals were in or outside of buildings. In some of our larger cities population density is as high as 145,000 per square mile in the daytime. This may drop to less than 50 per cent of this figure at night. By comparison the population density of Hiroshima was 35,000 per square mile. The Hiroshima bomb destroyed 4.7 square miles.

Various newspaper estimates of casualties in an American city in event of an atomic attack vary from 50,000 killed and 50,000 injured to 150,000 killed and 150,000 injured. Suppose, for example, we take the middle figure of 100,000 dead and 100,000 injured (fig. 14). The injured is the group with which we would be concerned. Applying the British and the Texas City figures, we know that of this number 2.7 per cent, at least, will have some eye injury due to blast. Of these 2,700 eye injuries, about 31 per cent will be seriously injured, giving a total of 837 severe and 1,863 mild eye blast injuries. The total flash and secondary burn cases would be approximately 40,000, of which at least 10 per cent would probably have burns involving the eyelids or eyeballs requiring some type of treatment (fig. 15). The number of these who would be severely burned cannot at this time be

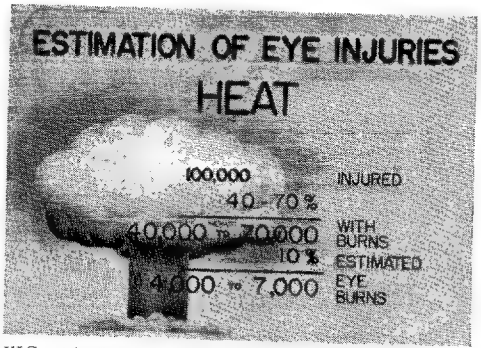

FIG. 15 Planning figures which might be used in estimating eye injuries produced by heat. These are admittedly only guesses since no accurate figures are available.

estimated. A total estimate is made, then, of 4,000 eyelid and eye burns requiring varying amounts of treatment.

Factors in Planning Care of Eye Casualties

Now suppose we are faced with this staggering total number of casualties. Who and what will be available to care for them?

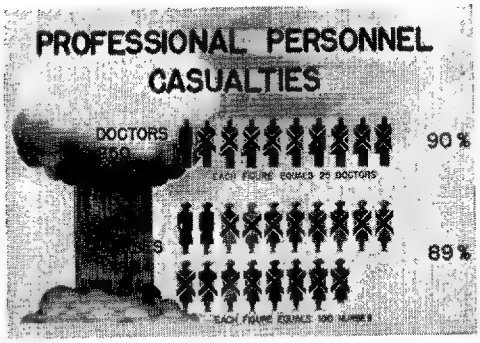

FIG. 16—Casualties which occurred in professional personnel at Hiroshima.

Personnel for care of casualties. The number of doctors and nurses would depend upon the time of day the explosion occurred. A higher percentage would probably be injured in a daytime attack on the heart of a city than at night. In Hiroshima, of about 250 doctors, 90 per cent were casualties and only 30 were able to work at the end of 30 days (fig. 16).

There were 1,780 nurses in Hiroshima before the bombardment. Of these, 1,654 were killed or injured (89 per cent casualties). This means, then, that personnel normally present to care for eye patients would not be available.

Hospital facilities. Hiroshima had 45 hospitals. Only three were left usable. Those within 3,000 feet were totally destroyed. Two were 5,000 feet away. They were of reinforced concrete construction and had 90 per cent casualties because of falling plaster, flying glass and fire. They could not be used for a long time. Hospitals between 7,000 and 10,000 feet distant were badly damaged and had many casualties.

Other facilities. In an atomic explosion, facilities other than hospitals would also be of immediate concern. Fires would break out immediately over a large area. Streets would be damaged, destroyed, choked with refugees trying to get out of the area, or blocked by fire. Telephone and other communication services would be interrupted. Electric power would not be available. The water supply would be cut off. At Hiroshima there were 70,000 breaks in the water supply system. The sewage system would also probably be destroyed.

SUMMARY

To summarize, then, the problem of proper eye care in the event of atomic attack:

1. There may be 100,000 total casualties with about 837 severely injured eye cases, 1,863 mildly injured eye cases, and 4,000 burned eye and eyelid cases of undetermined severity requiring immediate eye attention.
2. About 90 per cent of the ophthalmologists and eye nurses would themselves be casualties and, therefore, unable to help.
3. Hospitals within 3 miles would be incapable of being utilized. Their medical supplies would be destroyed in most instances.
4. Fires and falling buildings would be present in the damaged area, with streets blocked and filled with burning debris.
5. Electricity, water, food, communication and sewer systems would be damaged or destroyed.
6. It would be safe so far as residual radiation is concerned for rescue crews to enter the area if it was an air burst.

Any plan for the care of eye injuries which does not take into consideration these factors would be inadequate. The ideal treatment for eye injuries probably cannot be administered. The treatment which will accomplish the most for the largest number of cases in the hands of general practitioners and first aid personnel is probably the one that will do the greatest amount of good in the event of an atomic attack. If a good plan can be formulated for eye care it should result in better final vision for a larger number of people in the event of any future disaster.

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EMERGENCY EYE CARE IN DISASTER: THE LAYMAN WHO PRECEDES US

HEDWIG S. KUHN, M.D.
HAMMOND, IND.

COLONEL Byrnes has shown us that our concern with injured eyes in case of disaster (atomic or explosive) is mainly to be focused on the care of (1) burns—flash (which here means infra-red, ultraviolet and high intensity visible light) as well as burns from fire itself, (2) contusions and lacerations, (3) removal of foreign bodies, and (4) intraocular foreign bodies. Radiation as we think of it in this connection does not immediately concern the eyes. Its effects appear days, weeks, or even years after the disaster as keratitis or radiation cataract.

In Texas City almost all eye injuries occurred to office workers or business people watching the fire from behind windows and not wearing eye protective equipment; therefore, only 5 per cent of all injuries were eye injuries. In Britain during the blitz, as Colonel Byrnes' figures show, between 7 and 11 per cent of cases of serious injuries were eye injuries. If we use the low figure of 100,000 serious casualties in any major city where an atomic explosion occurs and use 10 per cent as the number having serious eye injuries, we have 10,000 serious eye injury cases on hand! This reiteration is necessary because we cannot realize the magnitude of these numbers. They remain fantastic and beyond comprehension. Dr. Ivy has been hammering the "facts of life" into academic heads for many weary months. Here, in brief, is the procedure:

Presented at the meeting of the Joint Committee on Industrial Ophthalmology, Oct. 10, 1950, Chicago, Ill.

Advance Roving First Aid Teams

1. Fill eye with 1½ per cent pontocaine in 1 to 3000 zephiran (packaging not yet decided on)
2. Tear a piece of sterile cloth from roll carried on back of first aid man and cover eye
3. Pain of an eye injury (other than burns) is not great, therefore hypodermic injection of morphine is not necessary.

First Aid Collecting Station

1. Cases of severe lacerations, perforations and obviously badly traumatized eyes
   a. In laceration of lids, leave same bandage on and send to the rear.
   b. Casualties with severe injuries of the globe should be sent to the rear *as stretcher cases* if at all possible. Bandage lightly if necessary.
   c. Tag everyone who has not already received preventive tetanus antitoxin so as to be sure that it is given at the next station.
   d. If sliver of foreign matter (glass, metal or wood) extends from eye, do not touch. Close eye with bandage and send to rear.
2. Burns in any one of the three categories (thermal, ultraviolet or chemical)
   a. Anesthetize again if necessary with pontocaine-zephiran solution.
   b. Insert hydrosulphosol (made up in new gelatin capsule form).
   c. Close securely with pressure bandage.
   d. Hypodermic injection of morphine if necessary.
3. Foreign bodies
   a. If on cornea, anesthetize (same solution of pontocaine and zephiran).
   b. To remove foreign body use applicator wound with cotton or closest substitute available.
   c. If very deep, close eye with some sort of patch and send to rear.

Casualty Station (may or may not have ophthalmologist)

1. *Careful inspection of eye*
   a. To further separate serious eye injuries overlooked in panic
   b. To pick up any unrecognized damage
   c. Lay personnel to use following findings as guides to serious damage:
      Severe pain in cases that are not burn cases
      Change in size or shape of pupil
      Blood in anterior chamber
      Protrusion of tissue of interior of eye
2. Treat and re-dress external eye burns, if necessary, with hydrosulphosol as used in collecting stations.
3. Dress minor lid lacerations
4. It is important to accept the use of medication approved by *local* community civil medical defense committee without argument or regard for personal preference. Disaster forces a condition resembling war; therefore we take orders just like in the Army.

Hospitals

Here is where the ophthalmologist takes over, but we must ever realize that we are still with limited means—a minimum selection of instruments, reduced supply of dressings, and a sharply reduced number of assistants. Our part, therefore, as ophthalmologists can be described as follows:

1. To assist directors of local (or regional) civil medical defense organizations; to implement and formu-

late the programs they set up with much more "give" than "take."

a. Accept previously agreed upon medication
b. Accept approved collecting station technics
c. Accept approved casualty station procedures
d. Accommodate to hospital needs

2. Assist those agencies set up to teach and train not only first aid but also self help, which is the one constructive good thing about this whole catastrophe defense effort.

a. Write simple instruction sheet to be inserted into loose leaf manual to be used by Red Cross and other citizens for teaching purposes.
b. Accept teaching assignments when asked or, better, volunteer.
c. Have ready *now* your own separate bag with *all* you need to work with for use at casualty station or at hospital so that you need not stand around and wait for someone to bring you instruments or supplies.
d. Offer your help and time now.

This whole gigantic potenial task is real and staggering, but it has been proved that (1) we *can* erect safeguards and prevent human destruction and human agony; (2) we *can* as physicians save lives and prevent permanent injury to legs, arms, and heads; (3) we *can* as ophthalmologists set up a properly trained lay "chain of command" and gear ourselves to the task of saving eyes also.

News - - - Notes

SECTION MEETINGS OF THE AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

The section meetings of the American Laryngological, Rhinological and Otological Society, Inc., will be held in January 1951.

The Eastern Section will hold its meeting on Friday, January 12, in the Bellevue-Stratford Hotel, Philadelphia.

The meeting of the Middle Section will be held in the Hotel Cleveland, Cleveland, Ohio, on January 15.

The Southern Section will meet at the Hotel Sans Souci, Miami Beach, Fla., on January 17.

The Western Section meeting will be held January 27 and 28 at the San Francisco County Medical Society Building, 2180 Washington Street, San Francisco.

AMERICAN ORTHOPTIC COUNCIL

The American Orthoptic Council will present its fourth annual intensive course in Orthoptics in Boston from July 5, 1951 through Aug. 31, 1951. Tuition is $150.00. The course is designed to present the necessary basic didactic instruction for students together with some practical instruction in order to prepare the student to complete a course of practical training and experience offered at various orthoptic centers over the country. The Council will make an effort to see that all students enrolled in the course have places to complete their practical training following the course. Applicants must be at least 18 years of age, have had a high school education and be sponsored by an ophthalmologist. A few scholarships are available to students from the Delta Gamma Fraternity Project. Inquiries and applications should be addressed to the American Orthoptic Council, Dr. Richard G. Scobee, 640 S. Kingshighway, St. Louis 10, Mo.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY

The third annual meeting of the Midwest Section of the Association for Research in Ophthalmology will be held at the University Hospitals, Iowa City, Iowa, on Monday, March 19, 1951. The program will consist of the presentation of scientific papers.

On the two days following the meeting, a symposium on "External Diseases of the Eye" will be presented by the Department of Ophthalmology, University of Iowa.

DELTA GAMMA FRATERNITY PROJECT: SIGHT CONSERVATION AND AID TO THE BLIND

The Delta Gamma Fraternity has a $1,500 annual fund and a $2,000 revolving loan fund from which smaller scholarship awards are available to those intending to become (1) orthoptic technicians, (2) teachers of partially seeing children, or (3) specialists for blind preschool children. Anyone wishing to specialize in one of these fields may be eligible for assistance, the amount in each case to be determined by the particular need and costs involved. For further information write to Mrs. Thomas Johnson, 1235 Longfellow, Detroit 2, Mich.

Applications for scholarships should be filed four months prior to the start of the desired course. Applicants with basic preparation in teaching, nursing, social work, nursery education, or related fields are eligible to apply. The scholarships are intended only for the courses directly relating to the field of sight conservation and aid to the blind. Candidates are selected with the advice of a professional committee which has as chairman, Dr. LeGrand H. Hardy of the American Orthoptic Council.

THE ISABELLA WILSON RESEARCH AND EDUCATIONAL FUND FOR FRONTAL SINUS PATHOLOGY

The American Academy of Ophthalmology and Otolaryngology has accepted the management of a special fund to be known as "The Isabella Wilson Research and Educational Fund for Frontal Sinus Pathology," initiated by Dr. Robert H. Fraser of Battle Creek, Mich. The pur-

pose of this fund is to pay the clerical help in review of case records from the office of Dr. Likely Simpson of Memphis, Tenn. The estate of Miss Isabella Wilson has contributed $250.00 to this fund, and contributions of not over $10.00 will be accepted from interested physicians and others.

OXFORD OPHTHALMOLOGICAL CONGRESS

The next meeting of the Oxford Ophthalmological Congress will be held in Oxford, July 5-7, 1951. The Doyne Lecture will be delivered by Dr. John Foster of Leeds. The two main discussions will be "Ophthalmology and Psychosomatic Medicine" and "The Modern Treatment of Retinal Detachment."

Inquiries should be addressed to Dr. Ian C. Fraser, Honorary Secretary, 12 St. John's Hill, Shrewsbury, Shropshire.

DISTRICT OF COLUMBIA

An exhibit honoring Hermann von Helmholtz, the inventor of the ophthalmoscope, will be displayed under the auspices of the Medical Museum of the Armed Forces Institute of Pathology and the Army Medical Library in the Armed Forces Medical Museum at Ninth and Independence Avenue, S. W., for six months beginning Dec. 17, 1950.

This date marks the hundredth anniversary of Helmholtz' letter to his father describing the instrument which was to enable doctors to see the interior of the human eye for the first time.

The Museum's extensive and comprehensive collection of ophthalmoscopes, which illustrates the development from the Helmholtz drawings to the modern electrified instrument, will be on display. Included will be the famous Dr. von Graefe's first instrument made by Sydow from the original drawing and given to the Medical Museum by Dr. Harry Friedenwald just before his death in the spring of 1950.

A rare Perrin three-dimensional ophthalmic atlas will also be on display. It was designed and manufactured about 1870 and extensively used at that early period to train students in ophthalmoscopy. Only three of these instruments are known to exist in this country.

The Army Medical Library has loaned many of the books to be shown. Included are many rare volumes, some of them found in the United States only at the Army Medical Library. Especially noteworthy in this respect are a Russian and two Japanese atlases of ophthalmoscopy, as well as the first printed illustration of the fundus of the eye, prepared by A. C. van Trigt for use in his doctoral dissertation soon after Helmholtz' report was made known, and the only published atlas of stereoscopic paintings of the fundus. A large number of contemporary American and foreign atlases will also be displayed.

ILLINOIS

The Chicago Laryngological Society held its first meeting of the season Nov. 6, 1950. The program was given by members of the department of otolaryngology of the Cook County Hospital in Chicago. It consisted of "Tracheotomy in Tetanus," by Drs. Emanuel Herzon and Edwin Killian; "Otogenic Intracranial Complications," by Drs. John Elsen, Elmer A. Friedman, and Norman Leshin; "Sarcoma of the External Auditory Meatus: Case Report," by Dr. Hans Von Leden; "Some Newer Surgical Considerations in Atrophic Rhinitis: Report of Work in Progress," by Drs. Maurice H. Cottle, Jack Allan Weiss, Edward F. Pottorff, and Emanuel Herzon.

The Chicago Ophthalmological Society held its regular meeting Nov. 20, 1950. At the afternoon session an instruction hour on "Orthoptics" was conducted by Miss Priscilla Allen, and a clinical program was presented by the departments of ophthalmology of Michael Reese and Mt. Sinai hospitals. The scientific program in the evening included "Report on the 16th International Congress of Ophthalmology," by Dr. Derrick Vail; "Optical Components of the Eye in Relation to Ametropia and Aniseikonia," by Dr. William F. Moncreiff; and "Macular Changes in Children from Maternal Rubella," by Dr. Samuel S. Blankstein.

Dr. Paul Hurwitz has been appointed to the faculty of the Chicago Medical School as assistant professor of ophthalmology.

The regular monthly meeting of the Chicago Laryngological and Otological Society was held Dec. 4, 1950. The pro-

gram consisted of "Experimental Studies of Negative Pressure Produced by Respiratory Cilia," by Dr. John J. Ballenger, department of otolaryngology, Northwestern University; and "Surgical Treatment of Carcinoma of the Esophagus," by Dr. Charles B. Puestow, department of surgery, University of Illinois.

KANSAS

Dr. William L. Benedict of Rochester, Minn., delivered the second E. J. Curran Lecture in Ophthalmology at the University of Kansas School of Medicine on December 14, 1950. His subject was "Differential Diagnosis of Exophthalmos."

LOUISIANA

The Tulane University of Louisiana School of Medicine announces a course in ocular pathology devoted to a study of tumors of the eye, adnexa, and orbit, to be given February 12 through 17, 1951. The fee for the course, which will be limited to an enrollment of 12, is $100. Assisting the regular staff will be Dr. C. S. O'Brien, Dr. John McGavic, and Dr. Theodore Sanders.

From February 19 through 24, 1951, a symposium on ocular pharmacology and therapeutics will be held at the Tulane University of Louisiana School of Medicine. Enrollment in the symposium will be limited to 150, and the fee will be $100. Drs. Alson E. Braley, Parker Heath, Irving H. Leopold, Robb McDonald, Frank W. Newell, and Alan C. Woods will assist the regular staff in conducting the symposium.

For further information regarding these courses, write to Dr. James H. Allen, 1430 Tulane Avenue, New Orleans, La.

MARYLAND

Dr. S. Rodman Irvine has been appointed associate professor of ophthalmology in the Johns Hopkins University School of Medicine for the year beginning October 1, 1950 and ending June 30, 1951.

MISSOURI

A meeting of the St. Louis Ophthalmic Society was held Nov. 16, 1950. The program consisted of "The Role of Hyperpyrexia in the Management of Eye Disease," by Dr. Bennett Y. Alvis, associate professor of clinical ophthalmology, Washington University School of Medicine; "The Treatment of Ocular Syphilis," by Dr. Leslie C. Drews, associate professor of ophthalmology, St. Louis University School of Medicine, and Dr. Gerald Barton (by invitation); and "Hyperphoria: Its Evaluation and Management," by Dr. Richard G. Scobee, assistant professor of ophthalmology, Washington University School of Medicine.

The Washington University School of Medicine announces a full time course in orthoptic technology. It will be given annually. The next course begins Sept. 17, 1951, and extends to June 15, 1952. Both didactic and practical training are given in orthoptic technology and a certificate is granted upon successful completion of the course. Tuition is $350.00. Facilities for instruction include the Motility Clinic of the Washington University Clinics and the St. Louis Ophthalmic Laboratory, the latter under the direction of Miss Anita Stelzer. The course is limited to eight students. Applicants must be at least 18 years of age and high school graduates, preferably with at least one year of college. Inquiries and applications should be addressed to Dr. Richard G. Scobee, Director of Graduate Training in Ophthalmology, 640 S. Kingshighway, St. Louis 10, Mo.

NEW JERSEY

The Section on Eye, Ear, Nose and Throat of the Academy of Medicine of Northern New Jersey met Nov. 13, 1950. Dr. Raymond E. Meek was guest speaker. He spoke on "Recent Advances in Ocular Surgery."

The Annual Clinical Conference of the Academy of Medicine was held at the Newark Eye and Ear Infirmary, Dec. 11, 1950.

The next meeting of the Section will be held March 12, 1951. Dr. James S. Shipman will be the guest speaker. He will present a paper on "Retinal Detachment and Some of Its Problems."

NEW YORK

The regular meeting of the Eastern New York Eye, Ear, Nose and Throat Association was held Nov. 2, 1950. It was "Clinic Day" and a number of interesting eye, ear, nose and throat cases were reported. The cases were described, discussed and coordinated by the guest, Dr. James W. Babcock, clinical professor

of otolaryngology at Columbia Presbyterian Medical Center, New York City.

At the evening meeting it was decided to cancel the December meeting, devote the January meeting to home talent, and hold the February meeting in Troy. Dr. Hecker gave a short resume of the E.N.T. highlights of the recent Academy meeting. Dr. Holohan then followed with a similar resume of the eye session there. Dr. Cetner presented a color sound film depicting an operation for "Tucking of the Superior Oblique Muscle." Dr. James W. Babcock showed a film and gave a paper on "The Effects of Streptomycin on the Labyrinth." He brought out the fact that persons taking streptomycin in large quantity lose the sensitivity of their labyrinths, have a positive Romberg, and cannot walk straight with their eyes closed. This drug has been used successfully in treating some cases of Meniere's syndrome. He described the Hallpike test. This treatment usually results in relief from vertigo in about 10 days, but leaves the patient dependent on his other senses for sense of position and motion. The paper was discussed by Drs. Volk, Hecker, Fierman, Cetner, Freund, and Sulzman.

PENNSYLVANIA

The thirty-fourth meeting of the Western Pennsylvania Eye, Ear, Nose and Throat Society was held at Indiana, Pa., Oct. 26, 1950. Dr. Raymond E. Jordan of Pittsburgh spoke on "Chronic Secretory Otitis Media," and Dr. William Linhart of Pittsburgh presented a paper on "Complications and Retinal Changes in Systemic Diseases."

Word has been received that Dr. F. B. Stevenson of Indiana, Pa., died Oct. 31, 1950. Dr. Stevenson was the organizer and first president of the Western Pennsylvania Eye, Ear, Nose and Throat Society.

A meeting of the Reading Eye, Ear, Nose and Throat Society was held Oct. 25, 1950, at the Wyomissing Club. A study club on "Ocular Therapeutics" was conducted. Instructors were Dr. James H. Parker of Reading and Dr. Joseph V. M. Ross of Berwick. Members who attended the Academy meeting in Chicago were invited to present their impressions of the highlights of the meeting. Reports on courses taken at the Chicago meeting were given by Drs. Ernest H. Dengler, Pottstown; C. Fremont Hall, Phoenixville; Fred R. Perfect, Wyomissing; Samuel A. Phillips, Allentown; Robert E. Shoemaker, Allentown; and Benjamin F. Souders, Reading.

The following officers were elected for the years 1950-51: president, Dr. Roy Deck, Lancaster; first vice-president, Dr. William J. Hertz, Allentown; second vice-president and president-elect, Dr. Harold L. Strause, Reading; treasurer, Dr. Philip R. Wiest, Reading; secretary, Dr. Benjamin F. Souders, Reading; and program chairman, Dr. John E. Keller, Reading.

The regular meeting of the Reading Eye, Ear, Nose and Throat Society was held Nov. 15, 1950. Dr. Harvey E. Thorpe, chief of the department of ophthalmology at Montefiore Hospital, Pittsburgh, spoke on "Management of Intraocular Foreign Bodies." A study club on the "Management of Early Deafness" was conducted by Dr. James E. Landis of Reading, and Dr. C. Fremont Hall of Phoenixville.

The Section on Ophthalmology of the College of Physicians of Philadelphia held a meeting Nov. 16, 1950. Dr. Parker Heath delivered the thirteenth annual de-Schweinitz lecture, "Tumors of the Iris: Pathology and Treatment"

POSITIONS AVAILABLE

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There is an opening in Washington, D. C., for a competent ophthalmologist to be associated with ophthalmologist in large private practice and with the George Washington University Medical School. The appointment comprises private practice and teaching, and research if so desired by the applicant. Application should be made to Mrs. R. McKenzie Ross, Room 2166 Department of Ophthalmology, George Washington University Hospital, Washington 7, D. C.

The Department of Otolaryngology and Oral Surgery at the University of Iowa Medical School will have four residencies available July 1, 1951. Applications should be submitted by Nov. 1, 1950, as the appointments are made Dec. 1, 1950. If you are interested, please write the Department of Otolaryngology and Oral Surgery, University Hospitals, Iowa City, Iowa, for information.

Listing of positions available should be sent to W. L. Benedict, M.D., 100 First Avenue Building, Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist; diplomate, resident, etc.); (2) type of position to be filled; and (3) whom to write for further information. Unless otherwise requested, listings will be published once only.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse or guarantee any of the published listings.

The staff of the Lewistown Hospital, Lewistown, Pa., is interested in having a man certified in ophthalmology locate in Lewistown. Lewistown has a population of approximately 16,000 and is located in central Pennsylvania. Lewistown Hospital serves a prosperous agricultural area with a population of approximately 75,000. New construction is under way which will give them a total of 186 beds plus 56 newborn bassinets. The medical staff consists of fourteen active members and thirty-three courtesy members plus two consulting and three honorary members. Write to Mr. Robert A. Kumpf, Administrator, Lewistown Hospital, Lewistown, Pa.

Otolaryngologist: A position for a resident is available at the Episcopal Hospital, Philadelphia, Pa. For details write Dr. Otto C. Hirst, Episcopal Hospital, Philadelphia 25, Pa.

A qualified ophthalmologist and otolaryngologist is needed in Denison, Texas, a city of 23,000 located on Lake Texoma. The area is industrial and agricultural. A high standard of living is maintained. There is no competition. Professional cooperation is assured if qualifications are adequate. For further information write Dr. Maurice A. Weisberg, Grayson X-Ray and Radium Clinic, Barrett Bldg., Denison, Texas.

DIRECTORY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETIES

INTERNATIONAL

INTERNATIONAL ASSOCIATION FOR PREVENTION OF BLINDNESS
President: Dr. P. Bailliart, 47 Rue de Bellechasse, Paris, France
Secretary-General: Dr. A. Churchill, 66 Boulevard Saint-Michel, Paris, 6, France

INTERNATIONAL COUNCIL OF OPHTHALMOLOGY
President: Prof. F. W. Nordenson
Secretary: Dr. Halger Ehlers, Rigshospital, Copenhagen, Denmark

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA
President: Dr. A. F. MacCallan
Secretary: Dr. F. Wibaut, P. C. Hoofstraat 145, Amsterdam, Holland

PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY
President: Dr. Conrad Berens
Secretary: Dr. Thomas D. Allen, 122 South Michigan Avenue, Chicago 2, Ill.
Time and Place: January 7-12, 1952. Mexico City

PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY, PUERTO RICO CHAPTER
President: Dr. Luis J. Fernandez, Box 2206, San Juan 10, Puerto Rico
Secretary: Dr. P. Fernandez
Place: San Juan

PAN-AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY
President: Prof. Justo Alonso
Secretary: Dr. Chevalier L. Jackson, 255 South 17th Street, Philadelphia, Pa.
Time and Place: Third Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology, Havana, Cuba, 1952.

FOREIGN

ALL-INDIA OPHTHALMOLOGICAL SOCIETY
President: Dr. G. Zachariah
Secretaries: Dr. S. N. Cooper and Dr. V. K. Chitnis, Laud Mansion, Queen's Road, Bombay 4, India

AUSTRIAN OTOLARYNGOLOGICAL SOCIETY, VIENNA
President: Prof. Dr. E. Schlander
Secretary: Doz. Dr. O. Novotny, Vienna IX, Alserstrasse 4, Austria
Time and Place: Annually

BELGIAN OPHTHALMOLOGIC SOCIETY
President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, Avenue Ruelens 179, Louvain
Time and Place: January, June and November. Bruxelles

BOMBAY OPHTHALMOLOGISTS' ASSOCIATION
Chairman: Rotated
Conveners: Dr. S. N. Cooper and Dr. B. D. Telang, Laud Mansion 21, Queen's Road, Bombay 4
Time and Place: 7:30 p.m., third Wednesday of each month. The seven ophthalmic hospitals of Bombay by rotation

BRITISH ASSOCIATION OF OTOLARYNGOLOGISTS
President: Mr. H. V. Forster
Honorary Secretary: Mr. F. C. W. Capps, 45 Lincoln's Inn Fields, London W. C. 2, England

BRITISH MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY
President: Mr. O. G. Morgan
Secretary: Mr. A. G. Cross, 27 Harley Street, London W. 1, England

CHENGTU OPHTHALMOLOGICAL SOCIETY
President: Dr. Eugene Chan
Secretary: Dr. D. S. Shen, Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China
Place: Eye, Ear, Nose and Throat Hospital, Chengtu, Szechuan, China

CHINESE OPHTHALMOLOGY SOCIETY
President: Dr. C. H. Chou
Secretary: Dr. F. S. Tsang, 221 Foochow Road, Shanghai

CHINESE OPHTHALMOLOGICAL SOCIETY OF PEKING
President: Dr. G. C. Lin
Secretary: Dr. H. L. Chen, Ophthalmological Dept., Peking University Medical School, Peking
Time: Bimonthly

DEUTSCHE OPHTHALMOLOGISCHE GESELLSCHAFT HEIDELBERG
President: Prof. Dr. med. K. Wessely
Secretary: Prof. Dr. med. E. Engelking, Heidelberg, Universitäts-Augenklinik

FACULTY OF OPHTHALMOLOGISTS
President: Dr. Frank W. Law, 45 Lincoln's Inn Fields, London, W.C. 2, England
Secretary: Dr. J. H. Doggart, F.R.C.S.

GERMAN OPHTHALMOLOGICAL SOCIETY
President: Prof. W. Lohlein
Secretary: Prof. E. Engelking, Heidelberg

HUNGARIAN MEDICAL TRADE UNION, SECTION OF OPHTHALMOLOGY
President: Prof. I. Csapody
Secretary: Dr. E. Galla, Krisztina Körut 139, Budapest, Hungary
Time and Place: Bimonthly. Second Eye Clinic, Budapest

HUNGARIAN OPHTHALMOLOGICAL SOCIETY
President: Prof. G. Horay
Assistant Secretary: Dr. Stephen de Grosz, University Eye Hospital, No. 1, Illesucca 15, Budapest

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Secretaries of societies are requested to furnish the information necessary to make this list complete and keep it up to date.

ISRAEL OPHTHALMOLOGICAL SOCIETY

President: Dr. Aryeh Feigenbaum
Secretary: Dr. E. Sinai, 9 Bialik Street, Tel Aviv

MIDLAND OPHTHALMOLOGICAL SOCIETY

President: Dr. F. A. Anderson
Secretaries: Dr. P. Jameson Evans, 51 Calthorpe Road, Edgbaston, Birmingham 15, England
Dr. R. D. Weeden Butler, 18 Highfield Road, Edgbaston, Birmingham 15, England
Place: Birmingham and Midland Eye Hospital, Church Street, Birmingham 3, England

NEDERLANDSCH OOGHEELKUNDIG GEZELSCHAP

President: Prof. A. W. Mulock Houwer
Secretary: Dr. T. A. Vos, Laan v. Meerdervoort 394, the Hague

NORTH OF ENGLAND OPHTHALMOLOGICAL SOCIETY

President: Mr. J. S. Arkle
Secretary: Mr. W. M. Muirhead, 70 Upper Hanover Street, Sheffield 3, England
Time and Place: October to May. Manchester, Leeds, Newcastle, Liverpool, Sheffield and Bradford

THE NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. D. M. MacRae
Secretary-Treasurer: Dr. E. I. Glenister, 513 Barrington Street, Halifax, Nova Scotia
Time: Four times yearly at dates to be arranged by the Executive

OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY OF ALBERTA

President: Dr. C. G. Elder
Secretary: Dr. M. R. Marshall, Wells Pavilion, University Hospital, Edmonton, Alberta, Canada

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA

President: Dr. Arthur H. Joyce
Secretary: Dr. Arnold L. Lance, 135 Macquarie Street, Sydney, New South Wales

OPHTHALMOLOGICAL SOCIETY OF COPENHAGEN

President: Prof. Dr. Holger Ehlers
Secretary: Dr. Carl Johan Moellenbach, Rigshospitalets oejenafdeling, Copenhagen O, Denmark
Place: Rigshospitalets oejenafdeling

OPHTHALMOLOGICAL SOCIETY OF EGYPT

President: Dr. Ibrahim Ahmad Mohammad
Secretary: Dr. Mahmoud Lutfi, Ophthalmic Hospital, Giza
Time and Place: Annual meeting during March. Summer meeting during August. Dar El Hekmah, 42 Kasr El Ainy Street, Cairo, Egypt

OPHTHALMOLOGICAL SOCIETY OF HOSPITAL DE NUESTRA SENORA DE LA LUZ

Chairman: Dr. Manuel J. Icaza y Dublan
Secretary: Dr. Jorge Meyran, Ezequiel Montes 135, México, D. F.
Time and Place: Second Friday of each month. Hospital de Neustra Senora de la Luz

OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND

President: Dr. L. S. Talbot
Secretary: Dr. W. J. Hope-Robertson, Kelvin Chambers, 16 The Terrace, Wellington
Time and Place: Annually. Auckland, Wellington, Christchurch and Dunedin alternately

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA

President: Dr. L. Staz
Secretary: Dr. J. Guillaume Louw, 901 Dumbarton House, Adderley Street, Cape Town, South Africa
Time: Annually

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

President: Mr. M. H. Whiting
Honorary Secretaries: Mr. A. G. Cross and Mr. A. J. B. Goldsmith, 45 Lincoln's Inn Fields, London W. C. 2, England
Time and Place: 1951 Congress of the Society, March 29-31, London

OXFORD OPHTHALMOLOGICAL CONGRESS

Master: Dr. F. A. Anderson
Hon. Secretary and Treasurer: Ian C. Fraser, F.R.C.S., Red Roofs, Kingsland, Shrewsbury
Time and Place: First week of July 1951, Oxford

PHILIPPINE OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Geminiano de Ocampo
Secretary-Treasurer: Dr. Carlos V. Yambao, Philippine General Hospital, Manila

POLISH OPHTHALMOLOGICAL SOCIETY

President: Prof. Dr. W. Kapuscinski
Secretary: Dr. S. Topolski, Piusa 38, Warsaw, Poland
Time and Place: Every two years—summer. Ophthalmic Clinic, Oczki 6, Warsaw

ROYAL SOCIETY OF MEDICINE, SECTION OF OPHTHALMOLOGY

President: Montague Hine, F.R.C.S.
Secretaries: Arthur Lister, F.R.C.S., 56 Wimpole Street, London, W.1., England
H. E. Hobbs, F.R.C.S., 129 Harley Street, London, W.1., England

SAO PAULO SOCIETY OF OPHTHALMOLOGY

President: Dr. Durval Prado
Secretary: Dr. Rubens Belfort Mattos, Rua B. Stapetininga, 29 F-3° andar, Sao Paulo, Brazil

SCOTTISH OPHTHALMOLOGICAL CLUB

President: Dr. E. H. Cameron
Secretary: Dr. John Marshall, 11 Clairmont Gardens, Glasgow, C.3, Scotland
Time and Place: Last Saturday of March and October, Edinburgh and Glasgow, in rotation

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

President: Dr. Diego M. Arguello
Secretary: Dr. Pedro F. Garcia Nocito, Vicente Lopez 1756 A, Buenos Aires, Argentina
Time and Place: Third Wednesday of every month. Buenos Aires

SOCIEDAD CHILENA DE OFTALMOLOGIA
President: Dr. Italo Martini
Secretary: Dr. Adrian Araya Costa, Renaca, no. 34, Santiago, Chile
Place: Santiago

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y DE OTORRINOLARINGOLOGIA
President: Dr. Jorge Suarez-Hoyos, Carrera 5a, no. 13-39, Bogota, Colombia
Secretary: Dr. Francisco Arango
Time and Place: Second Tuesday of each month. Club Medico

SOCIEDAD CUBANA DE OFTALMOLOGIA
President: Prof. Lorenzo Comas
Secretary: Dr. Heriberto Buch Granados, Calle B No. 668 ent. 27 y 29, Vedado, Habana, Cuba
Time and Place: First Thursday bi-monthly. Malecon No. 61 Bajos, Havana, Cuba

SOCIEDAD MEXICANA DE OFTALMOLOGIA
President: Dr. Teodulo M. Agundis
Secretary: Dr. Jose Luis Arce, Viena 3-5, Mexico D. F., Mexico
Time and Place: 8:30 p.m., first Tuesday of each month, Gral. PRIM 47

SOCIEDAD DE OFTALMOLOGIA DE CORDOBA
Chairman: Dr. Roberto Obregon Oliva
Secretary: Dr. Alberto Urrets Zavalia (hijo), 27 de Abril 255, Cordoba, Argentina

SOCIEDAD DE OFTALMOLOGIA DE GUADALAJARA
President: Dr. Elias Mendoza Gonzalez
Secretary: Dr. Jose Martin del Campo, Av. Juarez 211, Desp. 314, Guadalajara, Jal.
Place: Edificio Lutencia Despacho 101

SOCIEDAD DE OFTALMOLOGIA DEL LITORAL
President: Dr. Juan Manuel Vila Ortiz, Cordoba 1915, Rosario, Argentina
Secretary: Dr. Carlos M. Soto
Time and Place: Last Sunday in every month. Rosario

SOCIEDADE BRASILEIRA DE OFTALMOLOGIA
President: Dr. Natalicio de Farias
Secretary: Dr. Ismar Pereira, Praca Floriano 55, 5° andar, Rio de Janeiro, Brazil
Time: Third Friday of every month from April to December

SOCIEDADE DE OFTALMOLOGIA DEL NORTE
President: Dr. Jorge Luis Castillo
Secretary: Dr. Felix Berman, San Lorenzo 345, Tucuman, Argentina
Place: Mendoza 421, Tucuman

SOCIEDADE DE OFTALMOLOGIA DE MINAS GERAIS
President: Prof. Hilton Rocha
Secretary: Dr. Oswaldo Carvalho
Place: Belo Horizonte, Minas Gerais, Brazil

SOCIEDADE DE OFTALMOLOGIA E OTORINOLARINGOLOGIA DE RIO GRANDE DO SUL
President: Dr. Luiz Assumpcao Osorio
Secretary: Dr. Fernando Voges Alves, Caixa Postal 928, Porto Alegre, Rio Grande do Sul

SOCIEDADE DE OTO-RINO-LARINGOLOGIA DO RIO DE JANIERO
President: Dr. Aloysio Novis
Secretaries: Dr. Rubens Cabral, Rua Paissandu 73, Rio de Janeiro, Brazil
Dr. Mairelles Vieira

SOCIEDADE DE OFTALMOLOGIA E OTO-RHINO-LARYNGOLOGIA DE BAHIA
President: Dr. Theonilo Amorim, Barra Avenida, Bahia, Brazil
Secretary: Dr. Adroaldo de Alencar

SOCIETA OFTALMOLOGICA ITALIANA
President: Prof. Giuseppe Ovio
Secretary: Prof. E. Leonardi, Piazza degli Eroi, 11 Roma, Italy
Place: Roma Piazza degli Eroi 11

SOCIETE BELGE D'OPHTALMOLOGIE
President: Dr. L. Weekers
Secretary-General: Dr. M. Appelmans, 179 avenue Reulens, Louvain, Belgium
Time: Last Sunday of February, June and November

SOCIETE FRANCAISE D'OPHTALMOLOGIE
Secretary-General: Dr. Edward Hartmann, Hospital Lariboisiere, Paris 10, France

SOCIETY OF SWEDISH OPHTHALMOLOGISTS
President: Prof. S. Larsson
Secretary: Dr. K. O. Granstrom, Sodermalmstorg 4, Stockholm, Sweden

SOUTHERN OPHTHALMOLOGICAL SOCIETY, ENGLAND
President: R. Lang, M.R.C.S.
Hon. Secretary and Treasurer: Nigel Cridland, D.M., D.O., 25 Craneswater Park, Southsea, England

NATIONAL

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. J. Mackenzie Brown
Executive Secretary-Treasurer: Dr. William L. Benedict, 100 First Avenue Building, Rochester, Minn.
Time and Place: October 14-19, 1951, Chicago

AMERICAN ASSOCIATION OF EYE, EAR, NOSE AND THROAT SOCIETY SECRETARIES
President: Dr. Kenneth L. Craft
Secretary-Treasurer: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.

AMERICAN LARYNGOLOGICAL ASSOCIATION
President: Dr. Gordon B. New
Secretary: Dr. Louis H. Clerf, 1530 Locust, Philadelphia 2, Pa.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.
President: Dr. Louis H. Clerf
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Building, Rochester 7, N. Y.
EASTERN SECTION
Vice-President: Dr. Harry P. Schenck
Secretary: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
MIDDLE SECTION
Vice-President: Dr. Clarence W. Engler
Secretary: Dr. Walter H. Theobald, 307 North Michigan Avenue, Chicago 1, Ill.
SOUTHERN SECTION:
Vice-President: Dr. Charles C. Grace
Secretary: Dr. James W. McLaurin, Raymond Building, Baton Rouge 6, La.
WESTERN SECTION:
Vice-President: Dr. Lewis F. Morrison
Secretary: Dr. Harold Boyd, 1136 West Sixth Street, Los Angeles, Calif.

AMERICAN MEDICAL ASSOCIATION, SCIENTIFIC ASSEMBLY, SECTION ON OPHTHALMOLOGY
Chairman: Dr. A. Ray Irvine
Secretary: Dr. Trygve Gundersen, 101 Bay State Road, Boston, Mass.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY
Chairman: Dr. James M. Robb
Secretary: Dr. Sam H. Sanders, 1089 Madison Avenue, Memphis 3, Tenn.

AMERICAN OPHTHALMOLOGICAL SOCIETY
President: Dr. John H. Dunnington
Secretary-Treasurer: Dr. Maynard C. Wheeler, 30 West 59th Street, New York 19, N. Y.
Time and Place: Eighty-seventh annual meeting, June 7-9, 1951, Greenbrier Hotel, White Sulphur Springs, W. Va.

AMERICAN OTOLOGICAL SOCIETY
President: Dr. Kenneth Day
Secretary: Dr. John R. Lindsay, 950 East 59th Street, Chicago 37, Ill.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY
President: Dr. George E. Shambaugh, Jr.
Secretary-Treasurer: Dr. Joseph W. Hampsey, 806 May Building, Pittsburgh 22, Pa.

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.
Chairman: Dr. Walter H. Fink
Secretary-Treasurer: Dr. James H. Allen, 1430 Tulane Avenue, New Orleans, La.

CANADIAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY
President: Dr. R. G. C. Kelly
Secretary: Dr. J. Clement McCulloch, 380 Medical Arts Building, Toronto, Ontario

CANADIAN OPHTHALMOLOGICAL SOCIETY
President: Dr. R. F. Nicholls
Secretary: Dr. J. F. A. Johnston, 174 St. George Street, Toronto, Ontario

CANADIAN OTOLARYNGOLOGICAL SOCIETY
President: Dr. W. J. McNally
Secretary: Dr. Jules Brahy, 361 Sherbrooke Street East, Montreal 18, Quebec

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS, INC.
President: Mr. Mason H. Bigelow
Secretary: Dr. Franklin M. Foote, 1790 Broadway, New York 19, N. Y.

REGIONAL

ARK-LA-TEX OTO-OPHTHALMIC SOCIETY
President: Dr. W. Griffin Jones
Secretary: Dr. Frank L. Bryant, 2622 Greenwood, Shreveport, La.
Time and Place: First Monday of month, October through May. Shreveport Club

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY
Chairman: Dr. Ogden D. Pinkerton
Secretary: Dr. John P. Frazer, 1133 Punchbown Street, Honolulu, T. H.
Time and Place: Third Thursday of each month (dinner meetings). Pacific Club, Honolulu

INTER-MOUNTAIN OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. Homer E. Smith
Secretary: Dr. George B. Ely, 115 East South Temple Street, Salt Lake City, Utah
Time and Place: Third Monday of each month, September through May. University Club, 136 East South Temple, Salt Lake City

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY
President: Dr. S. B. Carruthers
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.
Time and Place: May 14, 1951, Mississippi Gulf Coast

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY
President: Dr. Benjamin A. Sachs
Secretary: Dr. Garrett L. Sullivan, 101 Bay State Road, Boston 15, Mass.
Time and Place: Third Wednesday of each month, November through April. Massachusetts Eye and Ear Infirmary

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. Robert L. Goodale
Secretary-Treasurer: Dr. Burton E. Lovesey, 76 Bay State Road, Boston 15, Mass.
Time and Place: Quarterly. Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY
President: Dr. Augustus B. Dykman
Secretary-Treasurer: Dr. Howard P. House, 1136 West Sixth Street, Los Angeles 14, Calif.

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. William B. Hubbard
Secretary: Dr. Frank A. Ware, 514 Genesee Bank Bldg., Flint, Mich.
Time and Place: Second Tuesday evening of each month, except summer months. Bancroft Hotel, Saginaw

SIOUX VALLEY EYE AND EAR ACADEMY
President: Dr. James Reeder, Jr.
Secretary-Treasurer: Dr. W. P. Davey, 627-632 Frances Building, Sioux City, Iowa

SOUTHERN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. Alston Callahan
Secretary: Dr. Edley H. Jones, 1301 Washington Street, Vicksburg, Miss.

WISCONSIN-UPPER MICHIGAN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. James K. Trumbo
Secretary: Dr. G. L. McCormick, 650 South Central Avenue, Marshfield, Wis.
Time and Place: May 1951. Wausau, Wis.

STATE

ARKANSAS STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. C. G. Hinkle
Secretary: Dr. K. W. Cosgrove, 113 East Capitol Avenue, Little Rock, Ark.

CALIFORNIA MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION
Chairman: Dr. George F. Keiper, Jr.
Secretary: Dr. Maurice W. Nugent, 2007 Wilshire Blvd., Los Angeles 5, Calif.

COLORADO OPHTHALMOLOGICAL SOCIETY
President: Dr. J. Leonard Swigert
Secretary: Dr. James C. Strong, 227 Sixteenth Street, Denver, Colo.
Time and Place: Every third Saturday. Colorado Medical Center

COLORADO OTOLARYNGOLOGICAL SOCIETY
President: Dr. Guy W. Smith
Secretary: Dr. Terry J. Gromer, 110 Metropolitan Building, Denver, Colo.
Time and Place: 6:30 p.m., first Saturday evening of each month. Oxford Hotel, Denver

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. W. Jerome Knauer
Secretary-Treasurer: Dr. Charles C. Grace, 145 King Street, St. Augustine, Fla.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Lester A. Brown
Secretary: Dr. Braswell E. Collins, 701 Elizabeth Street, Waycross, Ga.
Time and Place: First Friday and Saturday of March. General Oglethorpe Hotel, Savannah, Ga.

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. E. W. Dyar
Secretary-Treasurer: Dr. M. S. Harding, 308 Hume Mansur Building, Indianapolis, Ind.

IOWA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Byron Merkel
Secretary: Dr. Carl A. Noe, 601-611 Highley Building, Cedar Rapids, Iowa

KANSAS STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. N. L. Francis
Secretary: Dr. W. D. Pitman, First National Bank Building, Pratt, Kan.

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
Chairman: Dr. J. E. Croushore (Otolaryngology)
Co-Chairman: Dr. J. C. Gemeroy (Ophthalmology)
Secretary: Dr. R. W. Teed, 215 South Main, Ann Arbor, Mich. (Otolaryngology)
Co-Secretary: Dr. F. B. Heckert, 1105 Bank of Lansing Building, Lansing, Mich. (Ophthalmology)

MICHIGAN TRIOLOGICAL SOCIETY
President: Dr. William D. Irwin
Secretary: Dr. V. E. Cortopassi, 324 South Washington Avenue, Saginaw, Mich.
Time: Second Thursday of November, December, March, April and May

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Malcolm C. Pfunder
Secretary-Treasurer: Dr. Frank Adair, 822 Lowry Medical Arts Building, St. Paul 2, Minn.
Time and Place: Second Friday of each month, November through May. Alternating, Minnesota Club, St. Paul, and Minneapolis Club, Minneapolis

MONTANA ACADEMY OF OTO-OPHTHALMOLOGY
President: Dr. W. L. Forster
Secretary-Treasurer: Dr. F. D. Hurd, Medical Arts Building, Great Falls, Mont.
Time and Place: Semi-annually. Summer, Bozeman; mid-winter, Diamond S Ranchotel, Boulder

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. G. M. Billings
Secretary: Dr. MacLean B. Leath, 529 North Main Street, High Point, N. C.
Time and Place: Annually, beginning second Monday in September.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Max Simons
Secretary: Dr. Richard S. Fixott, 1020 Southwest Taylor Street, Portland 5, Ore.
Time and Place: Third Tuesday each month September through May. Old Heathman Hotel, Portland

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Jay G. Linn
Secretary: Dr. Daniel S. DeStio, Highland Building, Pittsburgh 6, Pa.
Time and Place: May 17-20, 1951. Galen Hall, Wernersville, Pa.

PUERTO RICO MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Luis J. Fernandez
Secretaries: Dr. Ricardo F. Fernandez, P.O. Box 2206, San Juan 10, Puerto Rico (Ophthalmology)
Dr. Carlos E. Munoz MacCormick, P.O. Box 604, San Juan, Puerto Rico (Otolaryngology)
Time and Place: Bimonthly. Puerto Rico Medical Association Building

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. William M. Carpenter
Secretary-Treasurer: Dr. Roderick Macdonald, Rock Hill, S. C.

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Sam H. Sanders
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Building, Memphis, Tenn.
Time: Annually

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. V. R. Hurst
Secretary: Dr. John L. Matthews, 414 Navarro Street, San Antonio 5, Texas
Time and Place: December 1950. Dallas

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Charles A. Young
Secretary-Treasurer: Dr. Peter N. Pastore, Box 25, Medical College of Virginia Station. Richmond 19, Va.

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Garnett P. Morison
Secretary: Dr. Melvin W. McGehee, 425 Eleventh Street, Huntington 1, W. Va.
Time: Biannual

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. William F. Krone
Secretary: Dr. Francis J. Grant, 1224 Salem Avenue, Hillside, N. J.
Time and Place: 8:45 p.m., second Monday of the month. The Academy of Medicine, 91 Lincoln Park, South, Newark

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. R. Anderson
Secretary-Treasurer: Dr. A. L. Peter, 406 Akron Savings and Loan Building, Akron, Ohio
Time and Place: 6:30 p.m., first Monday in January, March, May and November. Akron City Club

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Lester A. Brown
Secretary-Treasurer: Dr. James T. King, 384 Peachtree Street N.E., Atlanta 3, Ga.
Time and Place: 7:30 p.m., fourth Monday of each month, October to May. Academy of Medicine

BIRMINGHAM EYE, EAR, NOSE AND THROAT SOCIETY

President: Each member, in alphabetical order.
Secretary: Dr. David A. McCoy, Woodward Building, Birmingham, Ala.
Time and Place: 6:30 p.m., second Tuesday of each month, September through May. Thomas Jefferson Hotel

BOSTON CITY HOSPITAL AURAL AND OPHTHALMIC ASSOCIATION

President: Dr. Joseph Nerbonne
Secretary: Dr. Benjamin Riseman, 41 Bay State Road, Boston 15, Mass.

BROOKLYN OPHTHALMOLOGICAL SOCIETY

President: Dr. Mortimer A. Lasky
Secretary-Treasurer: Dr. Louis Freimark, 256 Rochester Avenue, Brooklyn 13, N. Y.
Time and Place: 8 p.m., third Thursday in October, December, February and April. Medical Society of the County of Kings, 1313 Bedford Avenue

BUFFALO OPHTHALMOLOGIC CLUB

President: Dr. Arthur L. Bennett
Secretary: Dr. Herbert R. Reitz, 446 Linwood Avenue, Buffalo, N. Y.
Time and Place: Second Thursday of each month, October through May. Park Lane

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Harold R. Watkins
Secretary-Treasurer: Dr. Philip R. McGrath, 843 Jefferson Building, Peoria, Ill.

CENTRAL NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Cecil B. Hert
Secretary-Treasurer: Dr. James L. McGraw, 619 University Building, Syracuse, N. Y.

CHATTANOOGA EYE, EAR, NOSE AND THROAT SOCIETY

Chairman and Secretary: Dr. Willard H. Steele, Jr., 552 McCallie Avenue, Chattanooga, Tenn.
Time and Place: 6:30 p.m., fourth Thursday in each month, October through May. Mountain City Club

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

President: Dr. Oliver E. Van Alyea
Secretary: Dr. Lawrence J. Lawson, 636 Church Street, Evanston, Ill.

CHICAGO OPHTHALMOLOGICAL SOCIETY

President: Dr. J. Robert Fitzgerald
Secretary-Treasurer: Dr. Gail E. Soper, 636 Church Street, Evanston, Ill.
Time and Place: 7:30 p.m., third Monday of each month, October to May. Chicago-Illini Union, 715 S. Wood Street

CINCINNATI OPHTHALMOLOGIC CLUB

Presidium: Dr. Donald J. Lyle, Dr. Karl W. Ascher, Dr. Josef D. Weintraub
Secretary: Dr. Josef D. Weintraub, 715 Provident Bank Building, Seventh and Vine Streets, Cincinnati, Ohio
Time and Place: Second Wednesday night of month, November through May. University Club, Fourth and Broadway

CLEVELAND OPHTHALMOLOGICAL CLUB

President: Dr. G. Leslie Miller
Secretary-Treasurer: Dr. Webb P. Chamberlain, Jr., 7405 Detroit Avenue, Cleveland, Ohio
Time and Place: Second Tuesday of November, January, February and April. Statler Hotel, Cleveland

COLLEGE OF PHYSICIANS OF PHILADELPHIA, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Wilfred E. Fry
Clerk: Dr. M. Luther Kauffman, Medical Arts Building, Jenkintown, Pa.
Time and Place: 8:15 p.m., third Thursday of every month, October through April. College of Physicians Building

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. W. B. Wilkinson
Secretary: Dr. Claude D. Winborn, Medical Arts Building, Dallas 1, Texas
Time and Place: First Tuesday of each month. Melrose Hotel

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. J. H. McNamee
Secretary-Treasurer: Dr. H. H. Gurau, 213 Bankers Trust Building, Des Moines, Iowa
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Des Moines Club

DETROIT OPHTHALMOLOGICAL SOCIETY

President: Dr. Cecil Lepard
Secretary: Dr. Arthur P. Wilkinson, 947 Fisher Building, Detroit 2, Mich.
Time and Place: Second Monday of each month, November through April. Wayne County Medical Society

DETROIT OTO-LARYNGOLOGICAL SOCIETY

President: Dr. B. F. Glowacki
Secretary-Treasurer: Dr. John R. Birch, 1010 Maccabees Building, Detroit 2, Mich.
Time and Place: Third Wednesday of each month, September to May. Wayne County Medical Society Building

EAR, NOSE AND THROAT CLUB OF ST. LOUIS

Chairman: Dr. Bernard J. McMahon
Secretary: Dr. Harry N. Glick, 1504 Grand Boulevard, St. Louis, Mo.
Time and Place: Third Wednesday in November, January, March and May. University Club Bldg.

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

President: Dr. Byron H. Porter
Secretary-Treasurer: Dr. E. Martin Freund, 762 Madison Avenue, Albany 3, N. Y.
Time and Place: 8 p.m., first Thursday of month, October to June. Albany, Troy, Schenectady, rotating monthly

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. W. H. McKenzie
Secretary: Dr. C. Keith Barnes, 921 Neil P. Anderson Building, Fort Worth 2, Texas
Time and Place: 6:30 p.m., first Friday of each month, except July and August. All Saints' Hospital

HOUSTON ACADEMY OF MEDICINE, OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SECTION

President: Dr. Lyle Hooker
Secretary: Dr. Claude C. Cody, III, 1304 Walker Avenue, Houston 2, Texas
Time: Second Thursday of each month, October through June

INDIANAPOLIS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY

President: Dr. Edwin D. Dyar
Secretary-Treasurer: Dr. J. Lawrence Sims, 809 Hume Mansur Building, Indianapolis 4, Ind.
Time and Place: 6:30 p.m., second Thursday of each month, November to May. Indianapolis Athletic Club

KANSAS CITY SOCIETY OF O. O. R. L.

President: Dr. John McLeod
Secretary: Dr. James W. May, 1016 Rialto Building, Kansas City, Mo.
Time and Place: Third Thursday of the month, November through May. Hotel President, 14th and Baltimore

LONG BEACH EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Edmund D. Godwin
Secretary: Dr. James V. Keipp, 110 Pine Avenue, Long Beach 2, Calif.
Time and Place: 6:00 p.m., monthly, September through May. Seaside Memorial Hospital

LOS ANGELES OPHTHALMOLOGICAL SOCIETY

President: Dr. Deane C. Hartman
Secretary: Dr. Daniel B. Esterly, 104 North Madison Avenue, Pasadena 1, Calif.
Time and Place: First Thursday of each month, September through June. Los Angeles County Medical Society Building, 1925 Wilshire Boulevard

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Alden H. Miller
Secretary: Dr. Victor Goodhill, 2007 Wilshire Boulevard, Los Angeles 5, Calif.
Time and Place: 6:00 p.m., fourth Monday of each month, September through May. Los Angeles County Medical Association Building, 1925 Wilshire Boulevard

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OTOLARYNGOLOGY

President: Dr. Victor Alfaro
Secretary: Dr. Frasier Williams, 1228 North Irving Street, Arlington, Va.
Time and Place: 7:00 p.m., third Tuesday in October, November, March and May. Army and Navy Club, 17th and Farragut Square, N.W.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

Chairman: Each member, in alphabetical order
Secretary: Dr. Sam H. Sanders, 1089 Madison Avenue, Memphis, Tenn.
Time and Place: Second Tuesday in each month, September through May. Memphis Eye, Ear, Nose and Throat Hospital

MILWAUKEE OTO-OPHTHALMIC SOCIETY

President: Dr. J. P. Wild
Secretary-Treasurer: Dr. Howard High, 324 East Wisconsin Avenue, Milwaukee 2, Wis.
Time and Place: 6:30 p.m., fourth Tuesday of each month, October to May. Athletic Club

MONTGOMERY COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

President: Dr. Robert A. Bruce
Secretary-Treasurer: Dr. L. N. Shroder, 144 West Fourth Street, Greenville, Ohio
Time and Place: First Tuesday of November, February, March and May. Dayton Country Club

MONTREAL OPHTHALMOLOGICAL SOCIETY

President: Dr. Jules Brahy
Secretary-Treasurer: Dr. Leo S. S. Kirschberg, 1390 Sherbrooke Street West, Montreal, Que., Canada
Time: Second Thursday of October, December, February and April

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. N. B. Morris
Secretary-Treasurer: Dr. Herbert Duncan, Bennie Dillon Building, Nashville, Tenn.
Time and Place: Third Monday evening of month, October through May. James Robertson Hotel

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY

Chairman: Dr. Milton L. Berliner
Secretary: Dr. John M. McLean, 525 East 68th Street, New York 21, N. Y.
Time and Place: Third Monday of the month, October through May. New York Academy of Medicine

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

President: Dr. Samuel Gartner
Secretary: Dr. Leon H. Ehrlich, 211 Central Park West, New York 24, N. Y.
Time and Place: First Monday of the month, October to May. New York Academy of Medicine

NUECES COUNTY EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. N. Meador
Secretary: Dr. Rex C. House, 228 Medical-Dental Building, Corpus Christi, Texas
Time and Place: 7:30 p.m., second Monday of each month, October through May. Dusty's, 1739 South Brownlee Street, Corpus Christi, Texas

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. C. W. McClure
Secretary: Dr. Richard A. Clay, 416 Northwest 13th Street, Oklahoma City, Okla.
Time and Place: First Tuesday of each month. University Hospital, 800 Northeast 13th Street, Oklahoma City

OMAHA AND COUNCIL BLUFFS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

President: Dr. Thomas T. Smith
Secretary-Treasurer: Dr. G. T. Alliband, 1020 Medical Arts Building, Omaha, Neb.
Time and Place: Dinner meeting third Wednesday monthly, October to May. Omaha Club

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

Chairman: Dr. Alfred Cowan
Secretary: Dr. John W. Deichler, 301 South 21st Street, Philadelphia 3, Pa.
Time: First Thursday of each month, November through April

PHILADELPHIA LARYNGOLOGICAL SOCIETY

President: Dr. Thomas F. Furlong, Jr.
Secretary: Dr. John J. O'Keefe, 255 South 17th Street, Philadelphia 3, Pa.

PITTSBURGH ACADEMY OF MEDICINE

President: Dr. John S. Plumer
Secretary: Dr. Samuel D. Evans, 1501-2 Park Building. Pittsburgh 22, Pa.
Time and Place: 8:00 p.m., fourth Monday of each month, October through May. Pittsburgh Academy, 322 North Craig Street

PITTSBURGH OTOLOGICAL SOCIETY

President: Dr. Daniel S. DeStio
Secretary: Dr. George C. Schein, 634 Washington Road, Pittsburgh, Pa.
Time and Place: Bimonthly

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY

President: Dr. Frank H. Wanamaker
Secretary-Treasurer: Dr. Willard F. Goff, 432 Stimson Building, 1215 Fourth Avenue, Seattle 1, Wash.
Time and Place: Third Tuesday of each month. Medical-Dental Building, Seattle

READING EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. Roy Deck
Secretary: Dr. Benjamin F. Souders, 143 North Sixth Street, Reading, Pa.
Time and Place: Third Wednesday of each month, September to July (dinner meetings). Wyomissing Club

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. E. W. Perkins
Secretary-Treasurer: Dr. J. Warren Montague, 1001 West Franklin Street, Richmond 20, Va.
Time and Place: First Tuesday of January, March, May and October. Commonwealth Club

ST. LOUIS COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. Anderson C. Hilding
Secretary: Dr. James P. Tetlie, 626 Medical Arts Building, Duluth, Minn.
Time and Place: 6:00 to 8:00 p.m., preceding the monthly St. Louis County Medical Society meeting. St. Mary's Hospital, Duluth

ST. LOUIS OPHTHALMIC SOCIETY

President: Dr. J. M. Keller
Secretary: Dr. Benjamin Milder, 539 North Grand Avenue, St. Louis 3, Mo.
Time and Place: 8:00 p.m., fourth Friday of each month, October through April, except December. Elliott Auditorium, McMillan Hospital

SAN FRANCISCO COUNTY MEDICAL SOCIETY, SECTION ON EYE, EAR, NOSE AND THROAT

Chairman: Dr. W. E. Borley, 655 Sutter Street, San Francisco, Calif.
Secretary: None
Time and Place: Fourth Tuesday of each month, except July, August and December. San Francisco County Medical Society Building

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

President: Dr. Phil Greene
Secretary: Dr. Cornelius E. Hagan, Jr., 508 Old National Bank Building, Spokane, Wash.
Time and Place: Fourth Tuesday of each month, September through May. Paulsen Medical and Dental Building

SUPERIOR CALIFORNIA EYE CLUB

President: Dr. Theodore Holstein, 523 Medico-Dental Building, Sacramento 14, Calif.
Secretary: Dr. John Berg
Time: Every third Thursday, eight months of the year

TORONTO ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

Chairman: Dr. A. Lloyd Morgan
Secretary: Dr. Joseph C. Hill, 174 St. George Street, Toronto 5, Ontario, Canada
Time and Place: Second Monday night of November, January, February and March. Academy of Medicine, Toronto

WASHINGTON, D. C. OPHTHALMOLOGICAL SOCIETY

President: Dr. Thomas A. Egan
Secretary-Treasurer: Dr. Joseph Dessoff, 1726 Eye Etreet Northwest, No. 813, Washington, D. C.
Time: January 8, 1951, May 7, 1951, joint meeting with the Baltimore Ophthalmological Society in March 1951

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

President: Dr. C. E. Imbrie
Secretary-Treasurer: Dr. F. E. Murdock, 28½ West Scribner Avenue, DuBois, Pa.
Time: Biannually. Third Thursdays of May and October

WILKES-BARRE OPHTHALMOLOGICAL SOCIETY

President: Dr. Vincent Gallizzi
Secretary: Dr. Samuel T. Buckman, 70 South Franklin Street, Wilkes-Barre, Pa.
Time and Place: Last Tuesday of each month, October through May, except December. Luzerne County Medical Society Library, 130 South Franklin Street

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION THE AMERICAN MEDICAL ASSOCIATION

As of December 1, 1950

Aurex Model F
Aurex Model H
Mfr. AUREX CORP.
1117 N. Franklin St.
Chicago 10

Beltone Mono-Pac
Beltone Harmony Mono-Pac
Beltone Symphonette
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Mfr. BELTONE HEARING AID CO.
1450 W. Nineteenth St.
Chicago 8

Cleartone Model 500
Cleartone Regency Model
Mfr. AMERICAN SOUND PRODUCTS, INC.
2454 S. Michigan Ave.
Chicago 16

Dysonic Model No. 1
Mfr. DYNAMIC HEARING AIDS, INC.
1042 Atlantic Ave.
Brooklyn 16

Electroear Model C
Mfr. AMERICAN EARPHONE CO., INC.
10 E. Forty-third St.
New York 17

Gem Model V-35
Gem Model V-60
Mfr. THE GEM EAR PHONE CO., INC.
50 West Twenty-ninth St.
New York 1

Maico Atomeer
Maico UE Atomeer
Maico Quiet Ear Models G & H
Mfr. THE MAICO CO., INC.
21 N. Third St.
Minneapolis 1

Mears Aurophone Model 200
1947-Mears Aurophone Model 98
Mfr. MEARS RADIO HEARING DEVICE CORP.
1 W. Thirty-fourth St.
New York 1

Micronic Model 101 (Magnetic Receiver)
Micronic Model 303
Mfr. MICRONIC CO.
727 Atlantic Ave.
Boston 11

Microtone T-3 Audiomatic
Microtone T-4 Audiomatic
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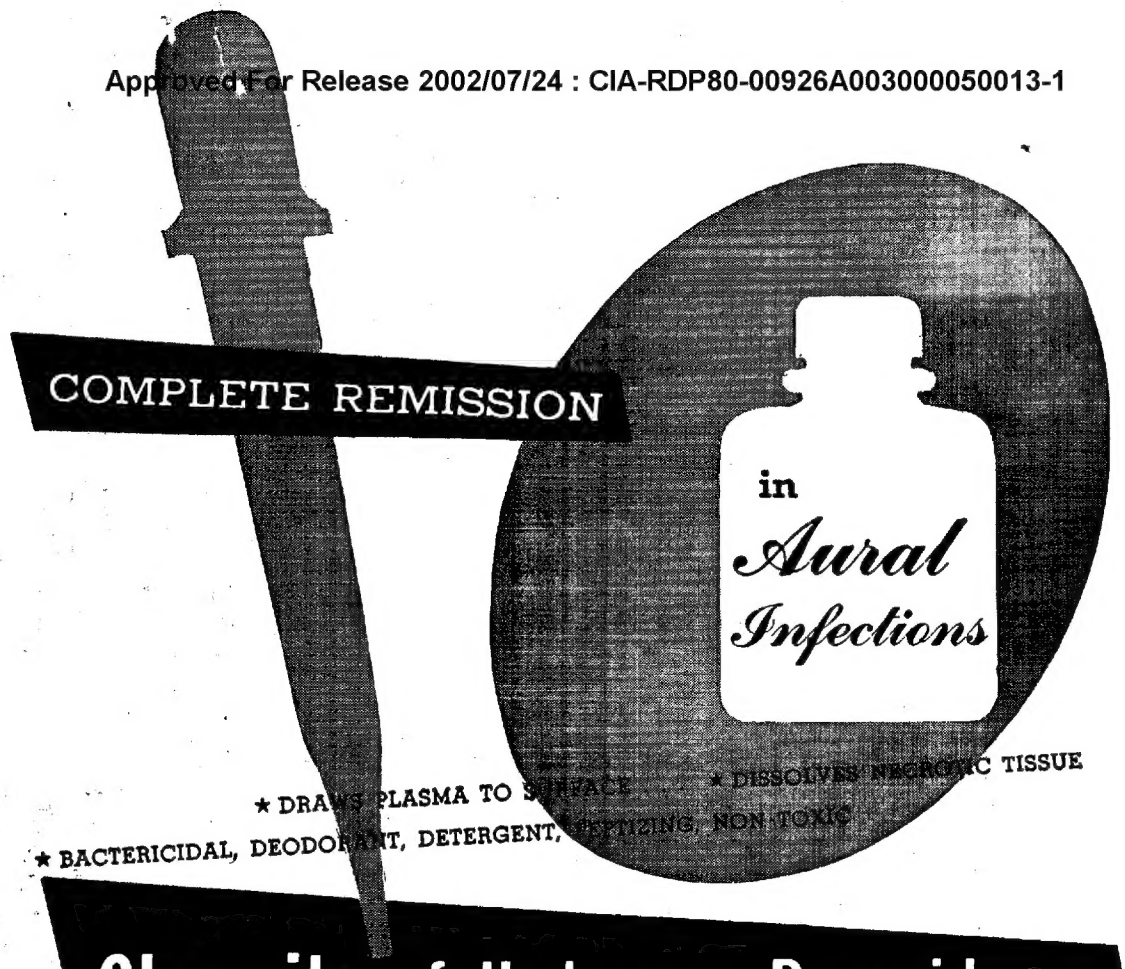

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